The American Journal of Surgery

EDITED BY

Thurston Scott Welton, M.D., F.A.C.S.

ORTHOPEDIC SURGERY · GYNECOLOGY

GASTROINTESTINAL SURGERY · OBSTETRICS

GENERAL SURGERY · ENDOCRINOLOGY

NEW INSTRUMENTS

Index to Volume XLIX *in This Issue*

September 1940

Special Monograph

The Diagnosis and Treatment of Hyperthyroidism and Associated Conditions

By ARTHUR GOETSCH, S.B., M.D.

THE AMERICAN JOURNAL OF SURGERY, Inc.

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Serum is an effective agent in combating all the effects of severe hemorrhage and resultant secondary shock except the loss of red blood cells, which is not serious unless very extensive. "Human Serum as a Blood Substitute," Levinson, S. O., et al., J.A.M.A. 114:6, 455, Feb. 10, 1940.

Plasma appears to be the ideal material for the reestablishment of proper circulation in secondary shock. . . . It is entirely safe and free from reactions and may be used in large and repeated doses. Plasma so prepared is ready for instant use.—"The Use of Citrated Plasma in the Treatment of Secondary Shock," Strumia, M. M., et al., J.A.M.A. 114:14, 1337, April 6, 1940.

Excessive hemoconcentration may be prevented by small repeated transfusions of Plasma.—"Plasma Transfusion in the Treatment of the Fluid Shift in Severe Burns," Elkinton, J. R., et al., Annals of Surgery 112:1, 150, July, 1940.

Plasma without cross matching is both safe and convenient. No reactions were observed when plasma, separated by centrifuging, was employed fresh or preserved either by refrigeration or by the lyophile process.—"The Intravenous Use of Serum and Plasma, Fresh and Preserved," Strumia, M. M., et al., Annals of Surgery, 111:4, 623, April, 1940.

Plasma should be an ideal substitute for whole blood in emergency treatment of shock and hemorrhage from war wounds . . . Plasma can be stored for a long period of time, and transported long distances safely.—"A Technique for the Preparation of a Substitute for Whole Blood Adaptable for Use During War Conditions," Tatum, W. L., et al., Military Surgeon, 85:6, 481, Dec., 1939.

Neutralization of agglutinins by both tissue and serum of the recipient also explains the safety of serum or plasma transfusions, without blood grouping and compatability tests.—"Suppression of Iso-Agglutinins and the Significance of the Phenomenon in Serum Transfusions," Lovinson, S. C., et al., J.A.M.A. 114:21, 2097, May 25, 1940.

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The Rationale of Suture Use

BRIEF DISCUSSIONS BASED ON REPORTS OF CURRENT RESEARCH AND CLINICAL OBSERVATION

Selection of Material for Buried Sutures

SOME difference of opinion has always existed regarding the choice of material for buried sutures in "clean" wounds. Discussion reached its peak during the early development of the absorbable suture and practically vanished from the literature as the efficacy of catgut became generally conceded. Its recent revival through the publication of numerous observations and reports gives rise to two questions (1) Have new and important discoveries been made? (2) Has there been a widespread change of opinion on this subject?

On the basis of present evidence the answer to both queries is negative.

Scrutiny of the reports on non-absorbable materials discloses little that is new either in behavior or methods of testing. The majority refer to silk and are accompanied by statistics indicating more favorable results with it than with catgut. With few exceptions, these statistics can be immediately classified as valueless since they compare fine sizes of silk with large sizes of catgut.

The net figures are unimpressive and, at best, demonstrate only the experience of the individual or institution reporting them. They may be further discounted by the fact that for every such experience others may be cited showing similar or better results in institutions using absorbable sutures exclusively. Also to be borne in mind are the potentialities in the presence of infection, the requirements as to special technic, and the contra-indications admitted by the staunchest advocates of non-absorbable materials.

On the absorbable side there has been less promiscuous writing and most of that published has been supported by more complete and thorough research. These writers have also had an advantage in that silk has been silk for centuries and little can be claimed for other non-absorbables beyond inertness and impermeability, whereas catgut has been subject to constant improvement. Moreover, the recent development of and trend to much finer sizes, 0 to 00000, is producing new figures showing markedly improved results.

All this, however, merely serves to confirm the contention of earlier writers that although catgut was amenable to improvement, most of the difficulty was due to the manner in which it was used and the quantity introduced.

As to current general opinion—the records of manufacturers and figures available from the hospitals themselves show a preponderance in favor of absorbable sutures and little change in the percentage during recent years. Scant significance may be attached to the volume of writing on either side since obviously there is a greater incentive to write on the minority opinion than on facts which are generally recognized and conceded.

CONCLUSIONS

1. The ideal buried suture has not as yet been developed. When it is, it will probably be absorbable since absorbability offers certain advantages in most circumstances.

2. In the meantime, the importance of the materials themselves is being greatly over-emphasized. Properly used, in comparable sizes and suitable circumstances, the difference in results is negligible.

3. Improvement in the surgeon's technic and a more logical application of sutures have a more direct bearing on the subject, and growing recognition of this fact is resulting in improvement in all branches of surgery.

Bibliography of the foregoing—prepared in collaboration with recognized authorities—will be supplied on request. DAVIS & GECK, INC., *Brooklyn, N. Y.*

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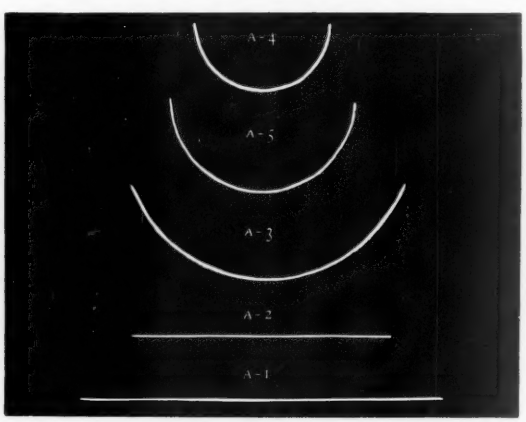

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1665	Black silk, B-1	18″	6-0, 4-0
1667	Plain–type A catgut, B-4	18″	3-0
1669	Mild chromic–type B, B-5	18″	4-0, 3-0
1669D	Mild chromic–type B, B-5	9″	4-0, 3-0

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1664	Black silk, B-1	12″	6-0. 4-0
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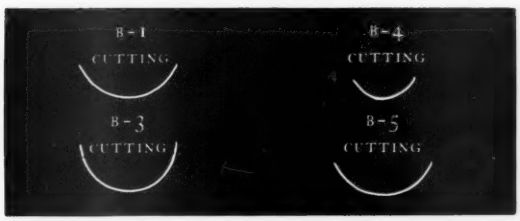

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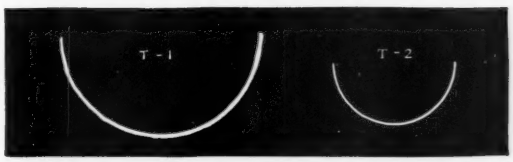

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1652	Kal-dermic, B-5	18″	4-0, 00
1653	Black silkworm gut, B-1	14″	4-0
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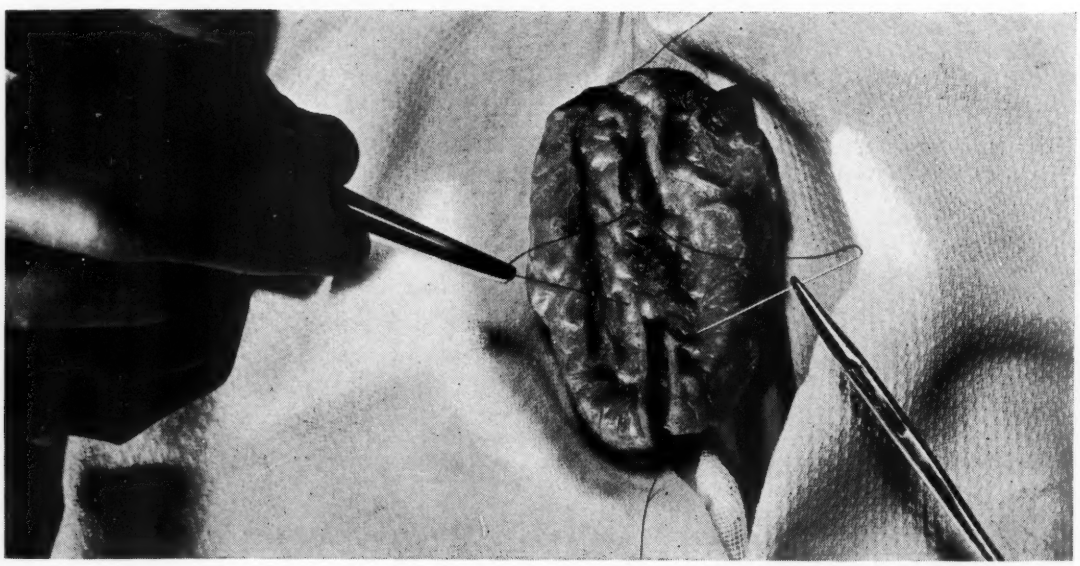

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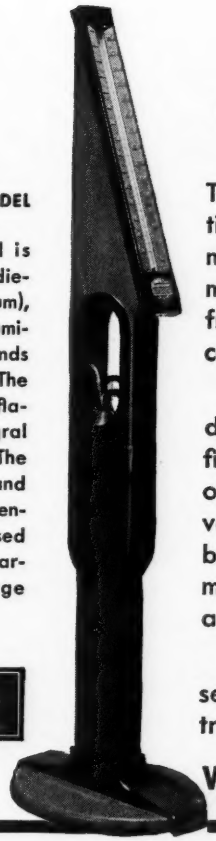

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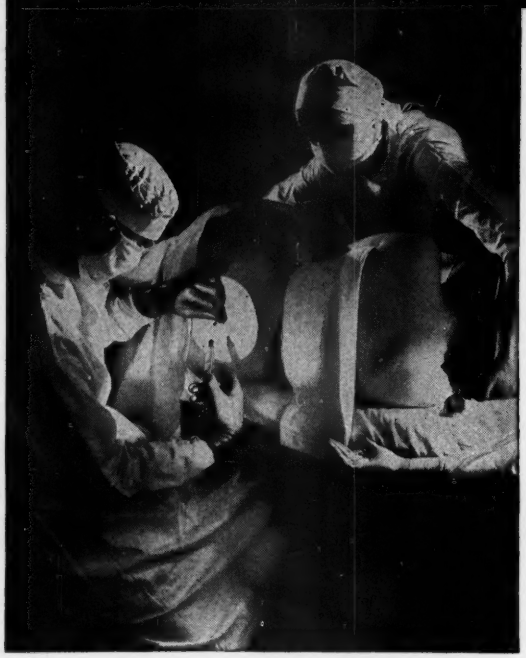
Basic factors
OF SUCCESSFUL SPINAL ANESTHESIA

of great importance - the duration of anesthetic action



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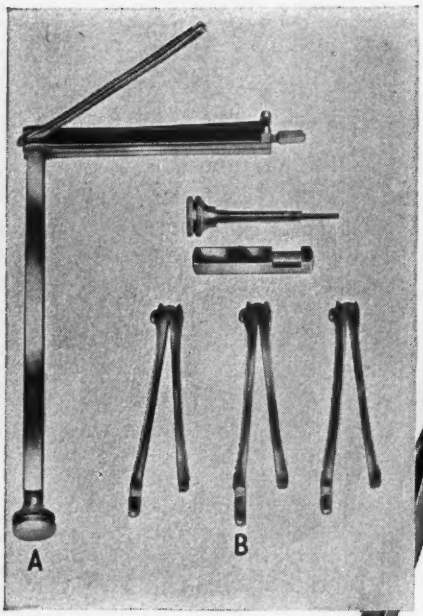

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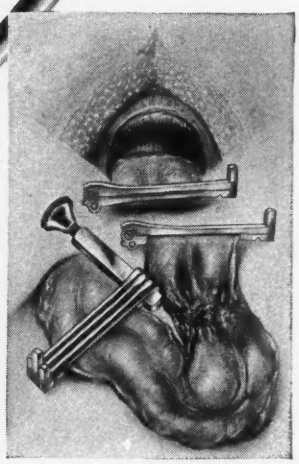

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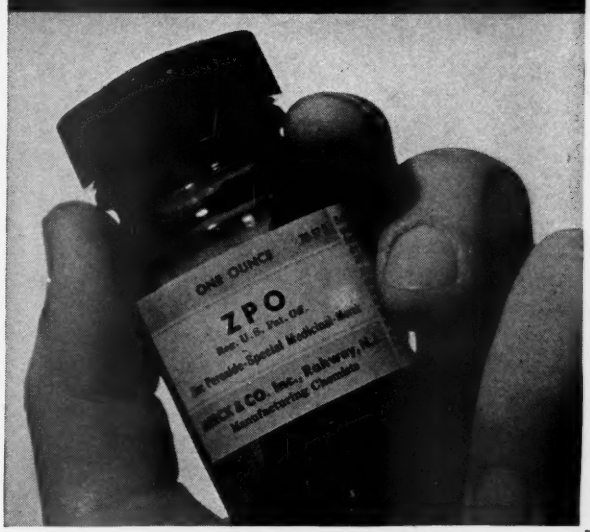

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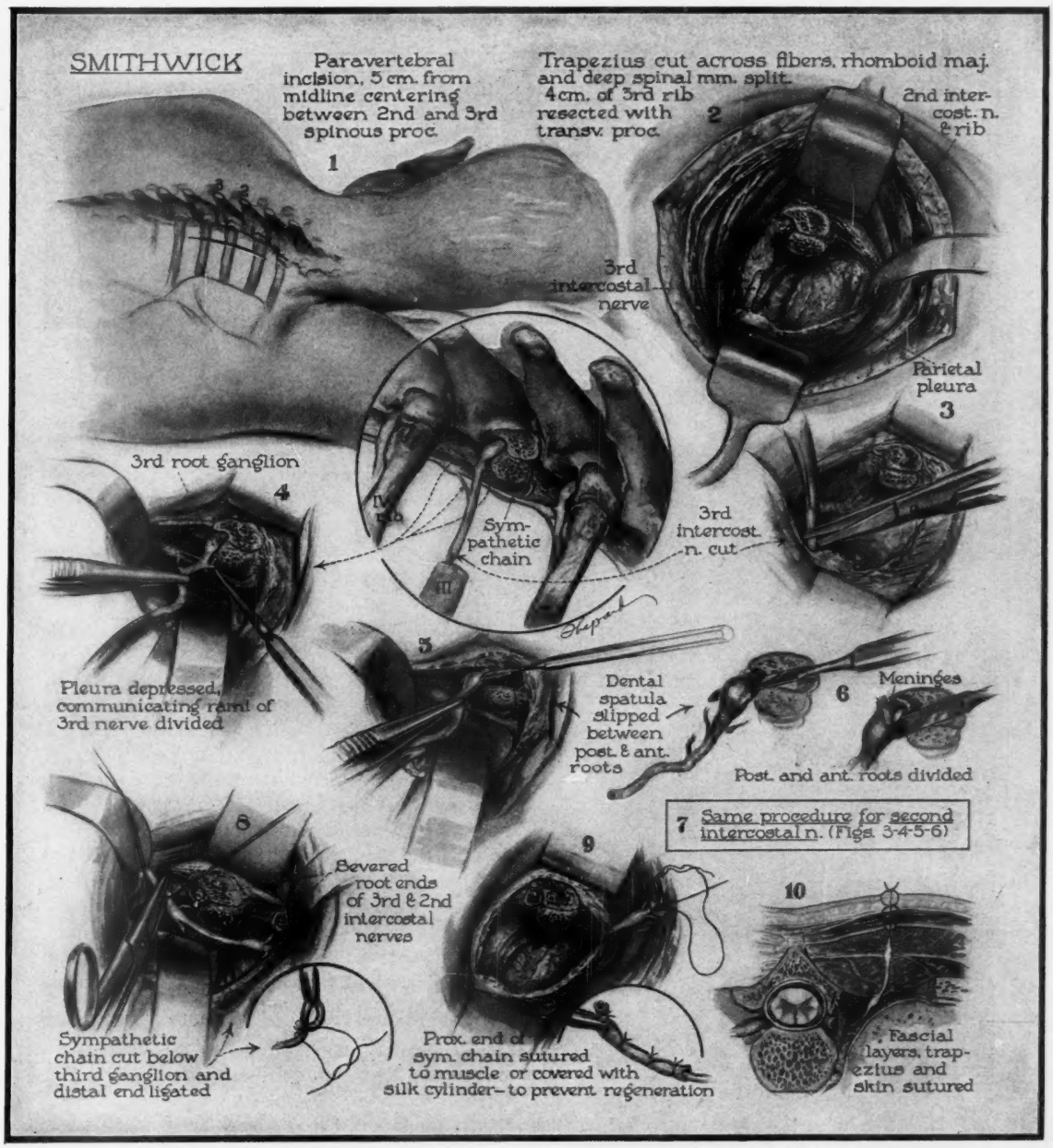
SMITHWICK
Paravertebral incision, 5 cm. from midline centering between 2nd and 3rd spinous proc.
1
Trapezius cut across fibers, rhomboid maj. and deep spinal mm. split. 4 cm. of 3rd rib resected with transv. proc.
2
2nd intercost. n. & rib
3rd intercostal nerve
Parietal pleura
3
3rd root ganglion
4
Sympathetic chain
3rd intercost. n. cut
Pleura depressed, communicating rami of 3rd nerve divided
5
Dental spatula slipped between post. & ant. roots
6
Meninges
Post. and ant. roots divided
7 Same procedure for second intercostal n. (Figs. 3-4-5-6)
8
Severed root ends of 3rd & 2nd intercostal nerves
9
10
Sympathetic chain cut below third ganglion and distal end ligated
Prox. end of sym. chain sutured to muscle or covered with silk cylinder—to prevent regeneration
Fascial layers, trapezius and skin sutured

OPERATIVE PROCEDURE—PLATE NO. 109—COPYRIGHT 1940, JOHNSON & JOHNSON

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OSLER AT OLD BLOCKLEY

NEW SERIES, VOL. XLIX SEPTEMBER, 1940 NO. 3

The American Journal of Surgery

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A PRACTICAL JOURNAL BUILT ON MERIT

EDITORIALS

"OSLER AT OLD BLOCKLEY"

DEAN CORNELL, noted American illustrator and mural painter, has finished the second of his proposed series of paintings of notable figures in American medicine. It is called "Osler at Old Blockley," and a reproduction is shown in the Frontispiece of this issue of the Journal.

This painting was first shown on the occasion of the dedication, on June eighth, of the Osler Memorial Building at The Philadelphia General Hospital. The old autopsy house was restored "As a Memorial to William Osler, M.D." by John Wyeth & Brother, Inc., of Philadelphia.

Osler came to Philadelphia as professor of clinical medicine early in October, 1884. He remained there for about five years. During that time Osler left an imprint so deep that years later those who worked with and under him resolved to build a shrine to his memory of the old "Post House" which was the scene of "The Great Physician's" inspiring teaching and work. Finally, this became a reality due to the timely assistance and generosity of John Wyeth & Brother, Inc.

Dean Cornell's painting depicts Osler addressing a group of residents at the bedside of a patient on the lawn under a large tree. The "Post House," where the painting will eventually find a permanent home, is shown in the background. Artist Cornell spent many months in historical research in preparation for the painting. The uniforms of the students and nurses were carefully authenticated. The grey pitcher, seen in the foreground, and other objects depicted were painted from originals of the period. The artist called upon many authorities for advice and criticism. We are told that one of the last conversations which the late Harvey Cushing had with persons, not members of his immediate household, was with Mr. Cornell concerning this contribution to American medicine.

The first painting of the series was "Beaumont and St. Martin." We told our readers about this in "The Artist Views American Medical Pioneers" (March, 1939). It has been exhibited at twenty-six medical schools during the past year and a half. It is still on tour as will be "Osler at Old Blockley."

These are truly epic works of art. We prize no end the framed reproductions of these paintings which John Wyeth & Brother so kindly presented to us. And we await with keen anticipation the next painting in the series by Dean Cornell who so ably has caught the spirit of the times and scenes depicted.

T. S. W.

DO THIS NOW

EARLY in July questionnaires from the A.M.A. began to reach doctors relating to classification of physicians for defense.

It goes without saying that this is a matter that should be answered at ONCE . . . IMMEDIATELY. When you receive this questionnaire, do not put it aside but fill it out without delay and return it to the A.M.A. The tabulation of the information received will take some time.

It is a professional and civic duty that this matter be awarded FIRST consideration in the day's business.

HOWARD L. UPDEGRAFF

HOWARD L. UPDEGRAFF, of Hollywood, California, widely known for his work in plastic and reconstructive surgery, died on August 8, at the age of forty-four years.

Dr. Updegraff was an associate editor of The American Journal of Surgery and gave valuable assistance in the form of constructive criticism, editorials and contributions. One of the last articles he wrote, entitled "Wound Closure, with Particular Reference to the Avoidance of Scars," will appear in our December issue.

Although young in years he played an important role in his chosen field and the horizon of his future accomplishments seemed far away. He was a real friend of the publisher and editor and they and his many friends regret this loss. All who knew him will miss him in the years to come.

ORIGINAL ARTICLES

FURTHER OBSERVATIONS ON THE TREATMENT OF BURSITIS OF THE SHOULDER*

ROBERT LEE PATTERSON, JR., M.D. AND RUSSEL H. PATTERSON, M.D.

NEW YORK CITY

IN reviewing the records of 104 cases of painful shoulders treated by us, the following diagnoses were found:

	No. of Cases
Bursitis	70
With calcification in the bursa or tendons	55
Without calcification	15
Periarticular inflammation	15
Sprain or subluxation	8
Arthritis	6
Rupture of the supraspinatus tendon	3
Congenital deformity	2

If we limit ourselves to the consideration of inflammation of the tendons about the shoulder and their overlying structure, the bursa, 80 per cent of these painful shoulders will be included.

Two years ago, one of us (R. L. P., Jr.) and Dr. William Darrach[1] reported sixty-three cases of bursitis of the shoulder treated by needle irrigation. According to the technique described, (Fig. 1) the patient is placed face upwards on a table. The skin over the affected area is prepared with iodine. One per cent novocaine is injected with a hypodermic needle into two areas: the first, anteriorly two fingerbreadths lateral to and on the same horizontal line with the coracoid process; the second, posteriorly about one fingerbreadth below the outer tip of the acromion just over the greater tuberosity of the humerus. After the skin is anesthetized an 18 gauge, 2½ inch needle is inserted into each area and is pushed gently down about ½ inch. If the bone is felt the needle is withdrawn about ⅛ inch. Here the needles should be in the bursa. Four to 10 c.c. of novocaine are used to get the flow between the needles started and 40 to 60 c.c. of normal saline are used to wash the bursa clean. The needles are removed and a dressing applied. Since this technique was reported, we have continued to use it. We are convinced that the procedure has a definite place in the treatment of bursitis because it gives in most cases a quick relief from severe pain and shortens the period of economic disability. With increasing experience, we have reached certain further conclusions.

Anatomy. The subdeltoid or subacromial bursa lies between the acromion and the deltoid muscle above, and the greater tuberosity of the humerus and the distal portion of the supraspinatus tendon below. It does not connect with the shoulder joint except when the supraspinatus tendon is torn.[2] Many of the bursae have septa dividing them into compartments. The function of this bursa is to prevent friction and to permit movement of the greater tuberosity in and out from under the acromion in motions of abduction and rotation. Any disease or injury which produces changes in this bursa disturbs its function and gives symptoms of bursitis, as for example: productive changes about the acromial process and tuberosity of the humerus incidental to arthritis; hemorrhage due to contusions about the shoulder; injuries of the tendons or capsule which actually tear the walls of the bursal sac, or repeated friction from calcium deposits in adjacent tendons.

Bursitis—a Secondary Disease. Primary inflammation, i.e., bacterial infection, does

* Read before the Orthopedic Section, New York Academy of Medicine, February 17, 1939.

occur. Hitzrot[3] reported three such cases. However, in none of our cases have we seen an infected bursa nor have we had an infection following irrigation or operation. It is therefore our belief that the common form of bursitis is not a primary disease but a secondary reaction in the walls of the bursa to some injury or pathology of the surrounding structures, especially those underlying the floor of the sac. Philip Wilson[4] has stated that the "all-embracing and commonly made diagnosis of subacromial or subdeltoid bursitis is usually inaccurate and rarely justified . . . that the great majority of the affections of the bursa are secondary to some primary lesion in one of the neighboring structures." Codman[5] since 1904 has continually stressed this important fact. Harbin[6] in 1929 brought out the importance of being able to distinguish a lesion of the tendon from one of the bursa.

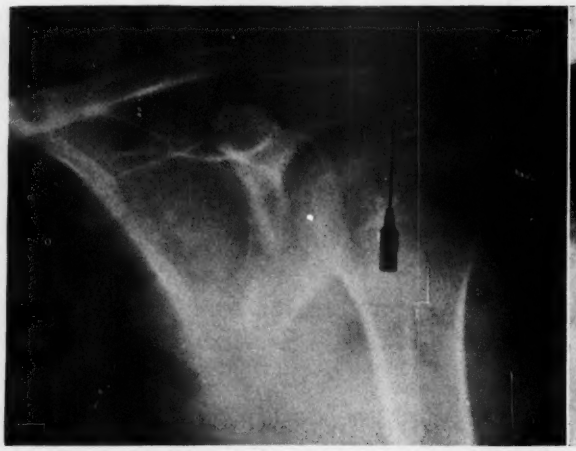

FIG. 1A. Needles shown inserted into calcium deposit in subacromial bursa. (From Darrach and Patterson.)

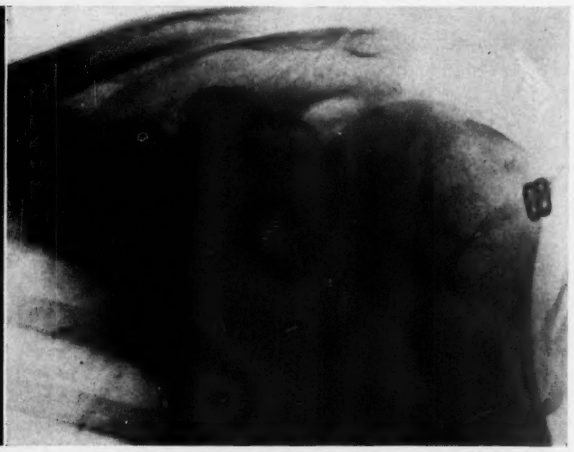

FIG. 1B. The same shoulder as in Figure 1A following irrigation. (From Darrach and Patterson.)

Calcium Deposits. In 78 per cent of our cases of bursitis calcium deposits were proved either in the bursa or in the tendon. When there is a definite history of injury sufficient to produce inflammatory changes in the tendons and capsule about the shoulder, we can readily account for the steps leading up to a calcium deposition. However, when there is no history of injury and yet calcification is present, it is difficult to account for what has happened. Our opinion is that wear and tear of usage profoundly affects that weakest and most susceptible structure about the shoulder, the supraspinatus tendon, whose function it is to anchor the head of the humerus against the glenoid so abduction can take place.[7] When man assumed the orthograde position, the additional weight of the arm made this function more difficult. The tendon has practically no blood supply and hence does not stand usage well. Over a period of years this process of wear and tear takes place, friction from above and below aiding, until calcification appears. This last step, according to Moschcowitz,[8] is nature's attempt to bring about repair in the presence of necrosis and a poor blood supply. Positive proof that this process has occurred is demonstrable by *x*-ray or by autopsy which, according to Codman,[9] reveals one torn tendon in every five examined.

Calcareous deposits occur not only in the supraspinatus, but also in the tendons of the teres minor, the infraspinatus, and the subscapularis muscles. We have observed and treated them in other parts of the body, such as the subtrochanteric bursa (Fig. 2), the semimembranosis bursa, and the radiohumeral bursa.

The calcareous deposit has been analyzed by several workers with varying results. It is usually reported between 11 per cent and 22 per cent calcium in the dry substance.

Calcium phosphate predominates over the carbonate. There is some magnesium.

These deposits may be present and yet sometimes shown apparently the same amount of calcium after irrigation as before and yet the patient's symptoms have dis-

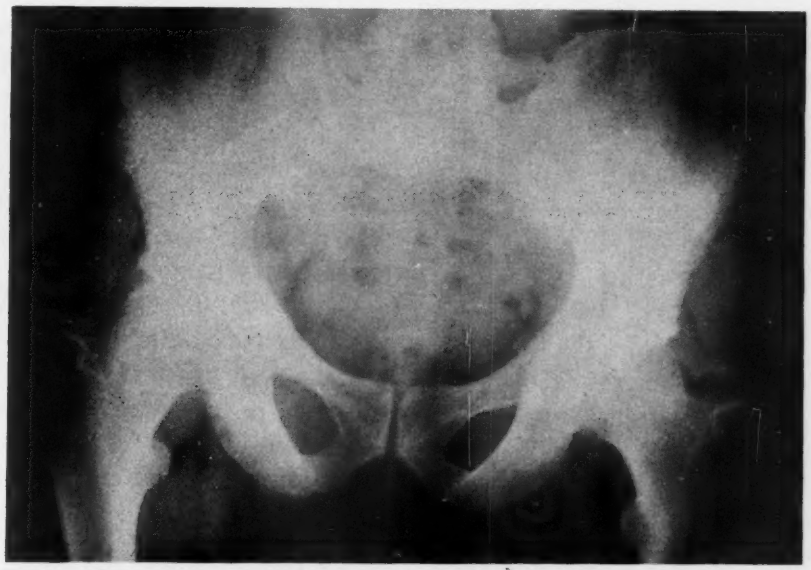

Fig. 2a. Represents calcified deposit about left hip. Acute onset with sciatic pain of two days duration. An example of the fact that calcification occurs about joints other than the shoulder.

give no symptoms, or they may slowly disappear over a period of time, whether treated by diathermy, by deep *x*-ray, or only by rest. The majority, however, remain or gradually increase in size until sudden trauma, sprain, or excessive friction occurs and rupture into the floor of the bursa takes place. Then acute symptoms develop.

An *x*-ray before irrigation or treatment is recommended, not only to determine the character and extent of the calcium deposit, if present, but also to make sure that some serious pathologic process, such as tuberculosis of the joint or a Brodie's abscess of the neck of the humerus, is not overlooked. However, we no longer make a point of taking an *x*-ray immediately after irrigation to see if the calcium has been completely washed out. If the patient's symptoms are relieved the presence or absence of calcium is relatively unimportant. A milky fluid, calcium in dilute solution, has been obtained in the irrigation of a bursa after a negative *x*-ray. We have relieved a patient's symptoms by irrigation without obtaining any demonstrable calcium. *X*-rays have appeared. Although we realize that the underlying process of tendinitis with calcification may return or may occur in other tendons about the shoulder and produce another attack of bursitis, we still maintain that, in irrigation, the relief of the acute pain and not the purely mechanical removal of the calcium deposit should be our aim.

X-ray Technique. In order not to overlook one of these deposits about the shoulder, we again want to stress the importance of taking *x*-rays in internal and external rotation. The former is easy since in the acute shoulder the patient's arm is adducted and internally rotated. But it is cruel and often impossible to rotate externally the acute shoulder. Now, as recently recommended by Lewis[10] at the Hospital for the Ruptured and Crippled, an oblique projection is taken. The patient stands with the shoulder against the cassette and the body at an angle of about 30 degrees.

The choice and selection of cases for irrigation is important. If we adhere to our original conclusions that the irrigation method is best for the acute case or the chronic case with an acute exaccerbation,

then we can be reasonably sure of the result.

Technique of Irrigation. 1. One should not hesitate to administer a general anesthetic, especially to a very sick patient or a very excitable one. We have used general anesthesia in some 2 per cent of our cases.

FIG. 2B. The same case as Figure 2A twenty-four hours after irrigation. Calcium has been washed out. Complete relief of symptoms occurred immediately.

2. Formerly, regardless of the points of tenderness, we inserted our needles in the position already shown. In the majority of cases the landmarks described will correspond to one of the tender spots. However, if neither of these two spots is the point of maximum tenderness, we now advise the insertion of one needle into the tender area and the second needle into either the front or the side position. Elmslie[11] stated in his excellent article that if a calcified type of lesion is present, a tender spot can always be found. One should not hesitate even to insert three or four needles if a good flow is not obtained between the first two, and if the tender spot has not been relieved.

3. We now think that a full range of motion should be obtained at the time of irrigation. When the needles have been inserted and the bursa washed, we remove the needles and gently put the arm through a full range of external rotation and abduction. If this cannot be done or if the patient complains too much,we reinsert the needles in the shoulder at the site of remaining tender spots. In other words, we are not satisfied until the patient has no pain and a satisfactory range of motion is possible.

Cases Not to Irrigate. We advise the irrigation of the acute shoulder, but there are cases of a borderline nature which are difficult. In five types of cases irrigation has been found to be certainly contraindicated:

1. The first type consists of patients with vague complaints of pain and numbness in the shoulder and arm, in whom there is a definite limitation of motion due to soft part contractures. Operative removal of the deposit, followed by manipulation and a long period of exercises has been found more advantageous in such cases.

2. The second type of case in which irrigation is to be avoided is the one with subjective symptoms of a vague nature but with no positive objective findings on examination. As a rule, this patient has had no conservative treatment and usually turns out to have something other than bursitis.

3. Irrigation has proved of little permanent value in cases in which *x*-rays showed a decreased width of the acromioclavicular joint, with roughening of the under surface of the acromion or of the tip of the tuberosity of the humerus, even though symptoms point to the bursa. By irrigating, we relieve tension in the bursa but do not change the underlying bony pathology. One possible exception to this statement is found in the patient with generalized arthritis who suffers bursal pain secondary to the bony changes. Irrigation produces temporary relief of pain and may help the patient to bear his generalized affliction.

4. There is a large group of cases classified as periarticular inflammation, second

in frequency in our series, which require an entirely different form of treatment. These patients are not usually seen by the specialist in the acute stages but are referred to him after the shoulder has failed to respond to all the routine forms of treatment—baking, massage, diathermy, plaster, injection, etc. Such cases have usually been overtreated and the patients have limited motion, complain of pain at night and on attempting to use the arms, and show atrophy of the deltoid and supraspinatus muscles. The skin over the shoulder has been "cooked" brown. Roentgenograms show only decreased density of bone. Certainly, irrigation of the bursa is not going to produce relief and would only add further strain. We now believe that most of these cases may be diagnosed as periarticular inflammation, i.e., tendinitis, capsulitis, myositis, etc., secondary to some general systemic infection or metabolic disturbance, such as toxemia, arthritis, gout, etc. Dickson and Crosley,[12] who reported 200 such cases, found a definite focus of infection in 176 of them. Out of the 200, 188 cleared up entirely, though it often took from one to six months. Our experience has taught the same.

Because the patient is sick, has a lowered resistance, and is physically tired of both pain and disability, it is imperative that all form of treatment be discontinued for a period of two weeks or longer. A vacation or rest from work is indicated. After this the following routine is recommended:

1. A complete physical examination with the removal of all possible foci of infection should be made and proper medication for sleep, diet, or anemia given.

2. The patient should be told that the process is a long one, often lasting four to six months, that perseverance is necessary, but that the results will be good with proper treatment.

3. Antigravity exercises should be performed regularly and efficiently each day.

4. Failure to respond to exercise after one month of treatment is an indication for admission to a hospital, where manipulation with breaking of adhesions should be performed. At the suggestion of Ober[13] we have injected the bursal region with 150 to 200 c.c. of saline and then manipulated the joint. This technique tends to dilute hemorrhage and to prevent the reforming of adhesions. We have tried it in a small number of cases and have found it to be of value. An abduction splint should immediately be applied and kept on for a period of three weeks. It should be removed daily, however, for exercises. In the average case, unless splinting is done following this treatment, the joint will very quickly "freeze" again.

5. Finally, do not irrigate, manipulate, or operate upon both shoulders at the same time when treating a bilateral shoulder condition. This is extremely important when handling cases of doubtful diagnosis and uncertain etiology.

SUMMARY

1. The most common condition found in the painful shoulder is bursitis.

2. Primary bacterial inflammation is rare in subacromial bursae.

3. Subacromial bursitis is nearly always secondary to injury of a neighboring structure.

4. Calcification can be demonstrated in about four out of five cases of bursitis of the shoulder.

5. Calcification in the five main tendons about the shoulder may eventually occur if they have been subjected to wear and tear or repeated small injuries.

6. The severity of the symptoms associated with bursitis is not in proportion to the amount of calcification present.

7. No treatment of a painful shoulder should be undertaken until *x*-rays have been studied. The oblique projection is strongly recommended.

8. Irrigation of the bursa is indicated in a large majority of acute cases. It is, however, definitely contraindicated in periarticular inflammation and all conditions of doubtful etiology.

9. Both shoulders should never be irrigated, stretched or operated upon at the same time.

10. The irrigation procedure is always best carried out in the hospital.

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A METHOD FOR PROTECTING THE SKIN IN "WINDOWED" UNPADDED CASTS*

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IT is often necessary to cut a window in unpadded plaster of Paris casts to change dressings or relieve pressure. Unless some method is used of replacing the even pressure afforded by the plaster splint, edema will appear with passive congestion of the underlying skin, and pressure necrosis may result. This is especially true when a Böhler's walking cast is being worn. The resulting pain and disability defeat the purpose of a walking cast by discouraging the patient to the point of not walking.

Due to the fact that it is not uncommon to see patients with areas of edematous skin bulging through cast windows, it was thought worth while to describe a method which prevents this condition from occurring.

The unpadded plaster of Paris splint is applied in the usual manner, and the area to be removed is outlined by an indelible pencil. A window of the proper size is cut, beveling the edges of the cast so that the smallest diameter is on the inner side next to the skin. It is not possible to use the portion removed as a plug for the window, for it is well frayed by the time it has been removed. The beveled edges of the cast are then lined with adhesive tape and the wound covered with five or six thicknesses of gauze. A strip of wet plaster of Paris gauze is wadded and packed into the open-

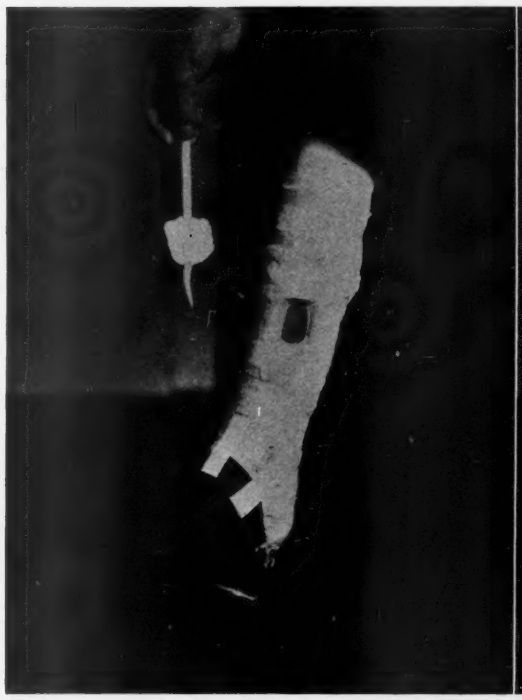

FIG. 1. Window in cast with beveled edges shown with removed plug. Although it is not apparent in the photograph, the plug fits accurately into the cast window.

FIG. 2. Window plugged with the plaster mould. It is necessary to encircle this area of the cast with a bandage, as walking loosens a strip of adhesive tape as shown above.

* From the Thomas-Davis Clinic.

ing and pressure applied until it hardens. It will be found that the plug of plaster will apply too much pressure to be comfortable, and layers of the gauze may be removed until the patient states that he is comfortable with weight bearing. The patient will rapidly learn how to pad the window and do his own dressing as often as needed. The plug may be shellacked to prevent its being softened by drainage. The tape lining and beveled edges make the plug easy to remove, and it is held by a bandage which envelops this portion of the cast.

THE fate of an injured hand is often decided promptly and permanently by the doctor who renders first treatment. What occurs in the first couple of hours can make the difference between a good or a bad result.
From—"Surgery of the Hand" by Couch (University of Toronto Press).

THE NEWER VENEREAL DISEASES*†

THEIR ASSOCIATION AND CONFUSION WITH NEOPLASTIC DISEASE

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IT is well that the agencies of man and of government have shouldered a responsibility to society in educating the public and particularly the physician to the problem of the venereal diseases. With the rapid advance and dissemination of our knowledge, particularly of the so-called newer venereal diseases, many a person with some venereal oddity, unresponsive to therapy, is at once labeled as suffering from granuloma inguinale, lymphogranuloma venereum or gangrenous chancroid with perhaps a superimposed fusospirochetosis. The physician has become so venereal disease-conscious when confronted with some singular genital lesion that he is oblivious to the probability of malignant disease.

The purpose of this paper is to draw attention to the frequency with which genital malignancy is clothed in the guise of a venereal disease. The malignancy may be concurrent, may be simulated or may be a direct sequel to the chronicity of one of the venereal diseases. Then again the newer venereal diseases, especially lymphogranuloma venereum, may manifest themselves as excessive, warty excresences; polypoid growths; elephantoid hypertrophy and hyperplasia of the vulvar tegumentary and subjacent connective tissue. The surface of such growths may be ulcerated. Granuloma inguinale and chancroid disease, particularly when a superimposed fusospirochetosis is present, may be so destructive and extensive as to simulate an ulcerative epithelioma. When one of these venereal diseases is mistaken for malignancy it is merely an error, but when a malignant process is diagnosed as a venereal infection and treated as such, it is indeed a sad and grave mistake.

The following cases are presented to emphasize certain of the points in question:

I. MALIGNANCY AS A SEQUEL TO VENEREAL DISEASE

CASE I. A white male, aged 33, was referred to the author with a progressive and destructive lesion of the penis. (Fig. 1A.) For two and one-half years he had received first, extensive antiluetic treatment, then a full course of potassium and antimony tartrate. Biopsies were taken on two occasions and examined by competent pathologists who reported them negative for malignancy. We found that repeated chancroid skin tests were positive, Frei tests and blood Wassermann and Kahn reactions negative. Stained films from the lesion revealed fusospirochetes and organisms resembling Ducrey bacilli. Biopsy now disclosed multiple foci of early epitheliomatous change (epithelioma in-situ). (Fig. 1B.) Radical operative procedures were undertaken with restitution to good health for six months. Then a recurrence appeared. Biopsy revealed an advanced type of epithelioma. (Fig. 1C.)

CASE II. A colored male, aged 28, is presented to compare the appearance and deformity caused by granuloma inguinale of seven years' duration. (Fig. 2.) Recrudescences have occurred with lapses of therapy. More recently *x*-radiation has improved the lesions considerably.

CASE III. A colored female, aged 49, has suffered with the various syndromes of lymphogranuloma venereum for seventeen years.

* Read before the Meeting of the American Public Health Association, Laboratory Section, October 18, 1939, Pittsburgh, Pennsylvania.

† From the Department of Obstetrics and Gynecology, Subdepartment of Experimental Medicine, University of Georgia School of Medicine. Aided in part by a grant from the U. S. Public Health Service to the University of Georgia group working on the newer venereal diseases, Dr. Richard Torpin, chairman.

Colostomy was performed eight years ago because of marked rectal stricture. (Fig. 3A.) At that time biopsy from the anorectal margin revealed chronic inflammation and granulation tissue. Condylomatous growths have appeared at the anal margin. (Fig. 3B.) Blood Wassermann and Kahn reactions, Frei and chancroid tests were positive. Biopsy taken from the same site revealed an epidermoid carcinoma and chronic inflammatory changes suggestive of venereal lymphogranuloma. The rectum was excised at autopsy and is shown in Figure 3A.

FIG. 1. A, epithelioma.

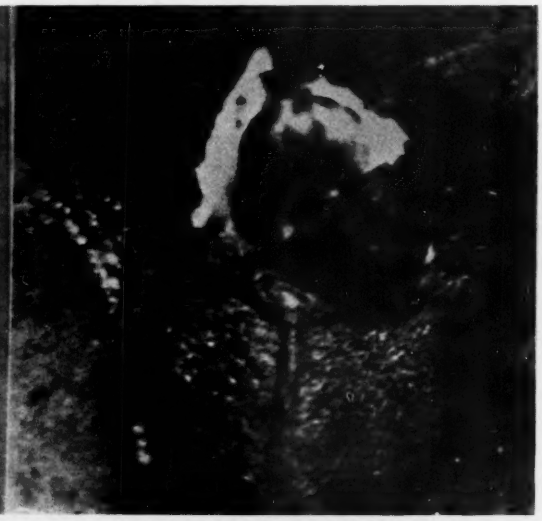

FIG. 2. Granuloma inguinale.

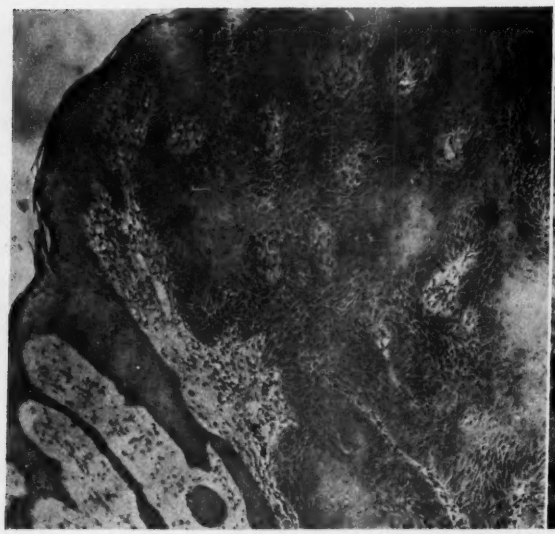

FIG. 1. B, photomicrograph (×150) epithelioma in-situ.

FIG. 1. C, photomicrograph (×150) advanced epithelioma.

CASE IV.* A colored female aged 23, had been hospitalized on several occasions for granuloma inguinale, lymphogranuloma venereum and pellagra. Extensive tartar emetic therapy resulted in eradicating the granuloma inguinale. The Frei test was positive. The labia, which recently had enlarged considerably, were covered by warty ulcerations and were excised.

The pathologic report by Dr. E. R. Pund is interesting: "Peritubular lymphocytic and plasma cell infiltration, dilation of lymphatics, fibrosis. Papillomatous hyperplasia of the epithelium. In several areas the epithelium is anaplastic and invades. Impression: Epitheliomas developing upon lymphogranuloma venereum." (Fig. 4.)

II. MALIGNANCIES MISTAKEN FOR VENEREAL DISEASES

CASE V. A white female, aged 50, was referred to the author as a case of lymphogranu-

* This case was previously reported by Pund, Greenblatt and Huie;[1] Cases I, III and IV were previously reported in greater detail by Cardwell and Pund.[2]

loma venereum of nineteen months' duration which would not respond to a variety of therapies. The lesion started as an indurated bubo in the left groin, which ultimately broke down. (Fig. 5.) Repeated Frei and chancroid tests as well as blood Wassermann reactions were negative. Biopsy revealed a squamous cell carcinoma.

Case vi. A male, aged 25, may be presented as a contrast to Case v. In many respects certain similarities in their marked ulcerative and destructive features may be noted. (Fig. 6.) However, this proved to be a destructive type of chancroidal infection of nine months' duration which responded rapidly to chancroid vaccine and sulfanilamide therapy.[3]

Case vii. A colored female, aged 55, was referred with a genital lesion diagnosed as lymphogranuloma venereum. There were elephantiasis of the labia, and marked induration and ulceration of the vagina. (Fig. 7.) Frei and chancroid tests were negative. Biopsy revealed squamous cell carcinoma.

A B

Fig. 3. A, rectal stricture of lymphogranuloma venereum. B, squamous cell carcinoma of anus.

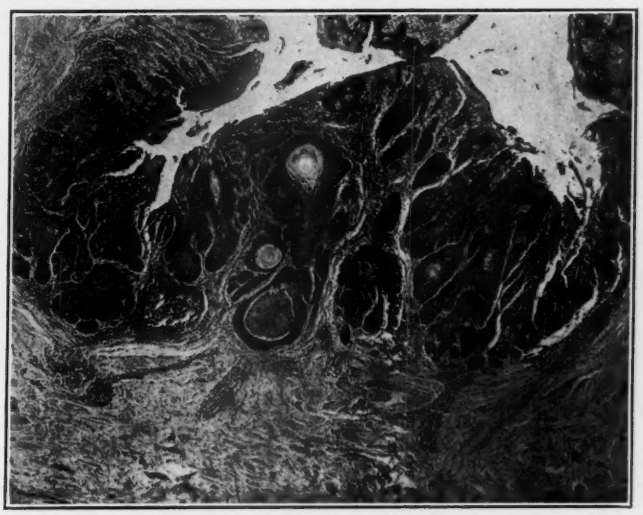

Fig. 4. Multicentric foci of epithelioma developing upon lymphogranuloma venereum. (From Pund, Greenblatt and Huie, in *Am. J. Syph., Gonor., & Ven. Dis.*, 22: 495, 1938.)

Case viii. A colored female of 19 years presented herself at the clinic with elephantiasis of the labia and ulceration of the vagina. (Fig. 8.) The Frei test was positive. This case of lymphogranuloma venereum is mimicked in a measure by the malignancy in Case vii.

Case ix. A colored female, 38 years of age, with a positive blood Wassermann reaction

and an ulcerative granulomatous growth about the anus had received antisyphilitic treatment for five months. (Fig. 9.) The lesion had been considered as syphilitic by her physician. Biopsy revealed a squamous cell carcinoma.

Case x. A colored female, aged 19, with mild elephantiasis of the labia and an anal syndrome marked by ulcerated growths, presented herself at the clinic. (Fig. 10.) The Frei test was strongly positive. Note the simularities of the anal lesions in this case of acute lymphogranuloma venereum and the carcinoma in Case ix.

Case xi. A colored male of about 35 years of age had received antiluetic treatment for eight months without benefit to an ulcerative lesion of the penis. (Fig. 11.) The chancroid test was positive, the Frei test negative and biopsy report indicated an advanced epithelioma.

Case xii. A white male, aged 19, was hospitalized with marked destruction of the glans penis. (Fig. 12.) Blood Wassermann and Kahn reactions, chancroid and Frei tests were negative, and only fusospirochetes could be found in fresh smears to account for the lesions. These healed promptly under therapy. (This case is included through the courtesy of Dr. Z. McDaniel.) Contrast this lesion with that of Case xi.

Case xiii. A colored male, about 40 years of age, was brought in for diagnosis by a public health physician from a neighboring state. The patient had received antiluetic treatment (because of a positive Wassermann) and when the lesion failed to respond, a course of Fuadin was given in the belief that the lesion might be granuloma inguinale. Chancroid and Frei tests

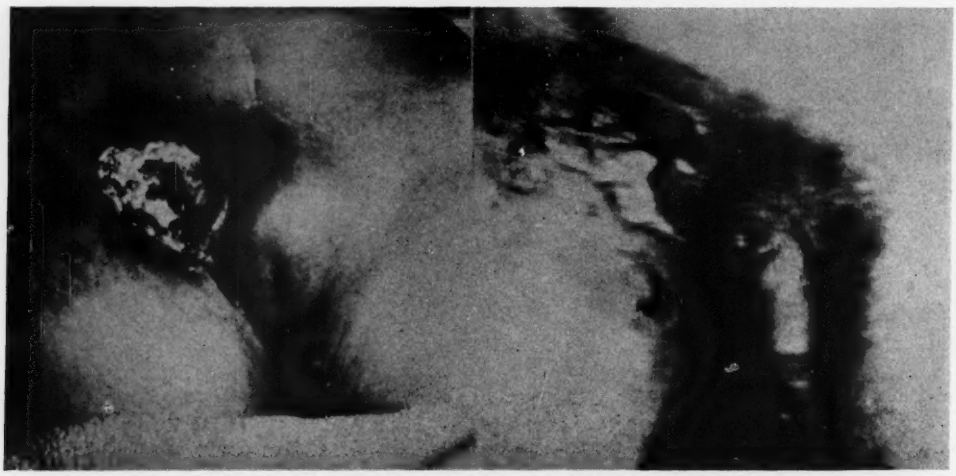

Fig. 5. Squamous cell carcinoma.

Fig. 6. Chancroid disease.

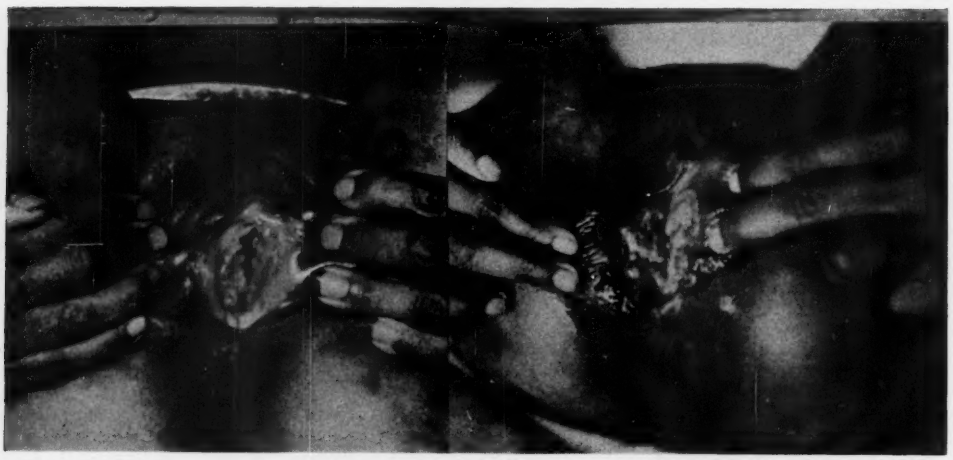

Fig. 7. Squamous cell carcinoma of vagina.

Fig. 8. Lymphogranuloma venereum with ulceration of vagina.

were positive. Biopsy revealed an advanced epithelioma (Fig. 13.)

Case XIV. In a colored male a chancroidal ulcer had sharply destroyed the whole of the glans. The clinical appearance was not unlike that of an epithelioma. (Fig. 14.)

Fig. 9. Epithelioma of anus.

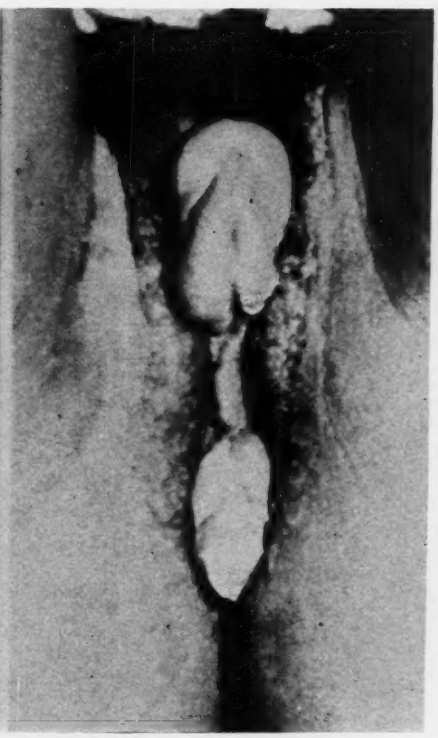

Fig. 10. Lymphogranuloma venereum (genitoanal syndrome).

Fig. 11. Epithelioma of penis.

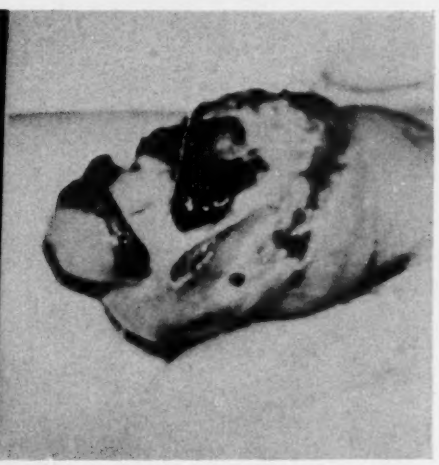

Fig. 12. (Primary?) fusospirochetal infection.

III. NEOPLASIA OF VENEREAL ORIGIN

Case XV. A colored female, aged 28, presented herself at the clinic with a pendulous mass, the size of a small orange, connected to the clitoris. The Frei test was positive and the chancroid test negative. (Fig. 15.)

Case XVI. A colored female, aged 30, presented herself at the clinic with massive growths involving the vulva and clitoris. A large papillomatous mass, 8 by 7 cm. in size, occupied the right lower vulvar region and perineum. The surfaces were covered by fine warty excrescences. (Fig. 16.) Ulceration was present within the vagina. Blood Wassermann and Kahn reactions, Frei and chancroid tests were all

positive. No improvement occurred under antiluetic treatment. The case finally proved to be one of lymphogranuloma venereum and granuloma inguinale.

growth extending from the lower right labium majus to the anus and another smaller one on the left labium majus. The surfaces were irregularly marked by grooved tracings and the

Fig. 13. Epithelioma.

Fig. 14. Chancroid.

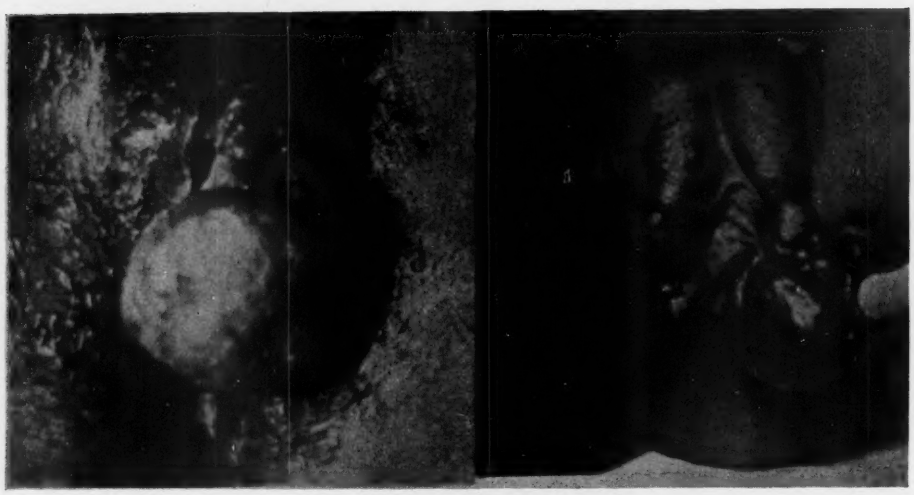

Fig. 15. Lymphogranuloma venereum.

Case xvii. A colored female, aged 28, was referred to our clinic for diagnosis with numerous papillary growths about the pudendal region. These were of many sizes and shapes and the colors varied from crimson to a violaceous hue. (Fig. 17.) Chancroid skin test was positive, Frei test negative. Biopsy report was condyloma accuminata.

Case xviii. A colored female, aged 22, presented herself with a massive firm tumor-like color was pink to pinkish gray. (Fig. 18.) Frei, chancroid and blood Wassermann reactions were all positive. Excision was performed and the histologic report was lymphogranuloma venereum and granuloma inguinale.

Case xix. Granular growths of the cervix, red and beefy in appearance, bleeding easily, are occasionally seen in our clinic. Figure 19 is a drawing of the cervix of a colored female, aged 33, who had been bleeding irregularly for three

months. A diagnosis of carcinoma of the cervix was made and radium applied. Biopsy report was granuloma inguinale. The pathognomonic relationship between ulcerative lesions of long standing, neglected through indifference of the patient or faulty diagnosis and

Fig. 16. Lymphogranuloma venereum and granuloma inguinale.

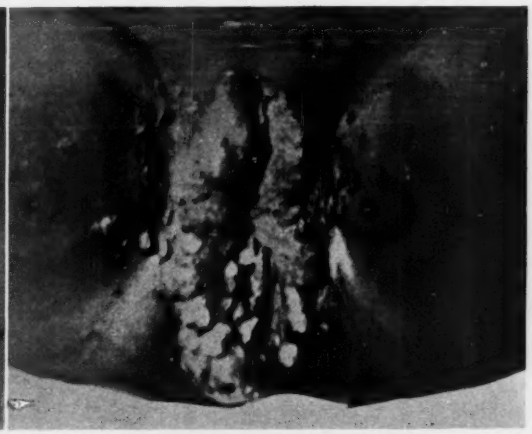

Fig. 17. Condyloma accuminata.

Fig. 18. Granuloma inguinale and lymphogranuloma venereum.

cells were numerous throughout the sections. There was no evidence of malignancy.

REVIEW OF LITERATURE

Cardwell and Pund[2] reported five cases in which venereal disease was complicated by the development of carcinoma on the involved sites. They suspected an etiologic maltreatment on the part of the physician. Liccione[4] noted the development of malignancy in two cases of inflammatory rectal stricture which were under direct observation. In spite of an extensive search of the literature he was unable to find this sequence of lymphogranuloma venereum and adenocarcinoma. David and Loring[5]

recently reported squamous cell carcinoma of the rectum in two proved cases of lymphogranuloma venereum with anorectal syndrome of long standing. Rosser, as well as Singleton, in the discussion of David and Loring's paper, refused to accept this sequence but preferred to lay blame on the fistulae which arose secondary to lymphogranuloma venereum as the cause of carcinoma. Lisa[6] reported one case of rectal lymphogranuloma venereum with a perineal fistula of ten years' duration in which a squamous cell carcinoma developed at the anus. Pund, Greenblatt and Huie,[1] in an evaluation of the rôle of the biopsy in the venereal diseases, found that epithelioma was superimposed upon one of the venereal diseases in four of the seventy-five biopsies studied. This led them to suspect a causal relationship. Reaves[7] drew attention to one case of granuloma inguinale and another where lymphogranuloma venereum of long standing was later accompanied by malignancy. Reaves suggested that chronic irritation over a long period of time is conducive to malignancy and that a "lookout for such pathology" is advisable.

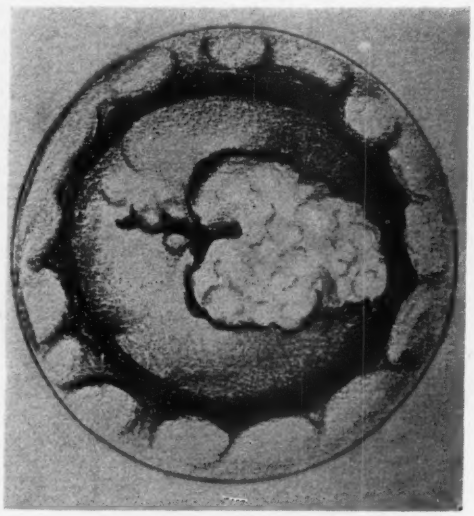

FIG. 19. Granuloma inguinale of the cervix.

DISCUSSION

The relation between persistent irritating chronic inflammatory processes and malignancy has long been known. It appears that certain individuals possess an epithelial integument that responds easily to irritant stimuli so that carcinoma may readily follow. This is apparent from a scrutiny of Cases I and III where the earliest neoplastic changes may be characterized as "epithelioma-in-situ." The persistent irritant in these cases was wrought by the chancroidal infection, with superimposed fusospirochetosis in one and the virus of lymphogranuloma venereum in the other. Biopsies in years past revealed nothing more than chronic inflammatory tissue. A biopsy retaken at the site some years later supplied evidence that malignant transformation had taken place.

Between granuloma inguinale and malignancy, the relationship is not so clear as in lymphogranuloma venereum. Few references are to be found in the literature. One case was reported by Cardwell and Pund[2] of granuloma inguinale of the cervix in which a malignancy was superimposed. Another case was reported by Reaves, as mentioned previously. On the other hand granuloma inguinale is frequently mistaken for malignancy clinically and not infrequently histologically. Briggs[8] mentioned that this disease is most commonly confused with that of lues but many cases treated for carcinoma have doubtless been venereal granuloma. Pund and Greenblatt[9] reported on two cases of granuloma inguinale of the cervix which were at first clinically mistaken for epithelioma. In fact in one of these cases the suspicion was so great that radium was applied at the time of biopsy. Histologic errors are made, as is implied in the report by Cannon[10] who twice presented cases which ultimately proved to be granuloma inguinale but were diagnosed as epithelioma by biopsy.

Randall, Small and Belk,[11] Gage,[12] and Pund and Greenblatt[13] in their studies on the histology of granuloma inguinale have warned against such an error. The following statement by the latter authors is perhaps representative, "The rete pegs become elongated and the resultant relationship so distorted that islands of keratinized epidermis are frequently found deep in the section. These give the appearance of epithelial pearl formation and may simulate

an early epithelioma." The ulcerative bubo as describe in Case v is perhaps unusual. Behcet[14] recently reported on a carcinoma of an inguinal gland which resembled lymphogranuloma venereum. In 1933 Favre[15] wrote on cancerous buboes, inflammatory in appearance which are simulated by lymphogranuloma venereum.

The presence of fusospirochetes in genital lesions[16] may result in marked destruction of tissue so that the lesion may simulate ulcerative and destructive types of epithelioma. Hyperplasia or neoplasia of venereal origin frequently presents problems in differential diagnosis. Cases x, xvi, xvii, xviii, and xix of our series are such examples. These at times may be excusably mistaken for malignant neoplasia.

A biopsy in such conditions is almost essential. Positive blood Wassermann reactions or Frei tests in themselves may prove a snare and a delusion by masking the true diagnosis of a malignant process. The rôle of the biopsy cannot be overemphasized, not only to rule out malignancy but as an aid in diagnosis. Proper histologic study may reveal treponema pallida, or the pathognomonic cell of granuloma inguinale or suggest the diagnosis of chancroid disease or lymphogranuloma venereum. It must be evident as an analysis of the cases reported show, that a positive Wassermann or a positive chancroid[17] or Frei test does not necessarily label the lesion in question and that biopsy can prove quite a resourceful undertaking.

SUMMARY

1. Too often benignant neoplasia of venereal origin has taken precedence, in the thoughts of the physician, over malignant neoplastic disease with dire consequences to the patient.

2. It must be stressed that an unusual genital lesion should cause the physician to be mindful that malignancy may exist either as a primary process, as a sequel to one of the venereal diseases, or concomitantly with one of them.

3. The importance of the biopsy and its interpretation in the differentiation of the venereal diseases by pathologists trained in this work cannot be overestimated. The biopsy must be placed side by side with such procedures as the blood Wassermann and Kahn reactions, the Frei and chancroid skin tests, if progress in diagnosis is to keep abreast with the great program for the public good by the sentinels of the public health.

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THE MANAGEMENT OF THE BREECH POSITION*

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MANY excellent papers have been written on the statistics and management of the breech position. The reason for the writing of yet another paper is merely to emphasize a few important points which would materially diminish the number of babies lost.

One hundred and three consecutive cases of breech extraction were performed. Three cases of twins are excluded because the weight of the individual children was less than 5½ pounds.

TABLE I
PARITY

Para 0	76
Para 1	16
Para 2	4
Para 3	2
Parity undetermined	5

There was no maternal mortality. The gross fetal mortality was eight, or 7.76 per cent. The corrected fetal mortality revealed four fetal deaths, or 3.8 per cent. Forceps were applied to the aftercoming head thirteen times. Peculiarly enough, the largest child extracted in this series (10½ pounds) did not require forceps on the aftercoming head; some of the other large infants were likewise born without such aid.

Complications. One child developed an Erbs palsy. This was a case in which the cord was prolapsed. The cervix was dilatable and a manual dilatation and rapid extraction had to be performed to save the baby.

One mother suffered a third degree tear which was immediately repaired and which was completely healed before she left the hospital.

Discussion of Fetal Mortality. CASE I. Delivery was very easy. The baby appeared weak and limp. Congenital lues was strongly suspected in this case.

CASE II. The cord was prolapsed and neither the family doctor nor I could hear the fetal heart prior to the commencement of the delivery.

CASE III. The mother had a premature separation of the placenta and the fetal heart could not be heard for some time prior to the delivery. She delivered spontaneously.

CASE IV. The mother had a contracted pelvis. When I first saw her she had been in labor for a long time and had had many vaginal examinations. There seemed to be a mild disproportion. I deemed it best under the circumstances to deliver naturally. Fetal death was most probably due to cerebral hemorrhage. I have since delivered this patient by cesarean section of two living children and each time the presentation was breech.

CASE V. Fetal heart could not be heard prior to delivery.

CASE VI. Macerated fetus.

CASE VII. Baby died about a week before onset of labor.

CASE VIII. Baby died about a week before onset of labor.

From even a brief perusal of this mortality table one can easily see that the results in this series could have been better if all the cases had been supervised throughout pregnancy and labor by an experienced obstetrician.

DISCUSSION

Our statistics can be improved if we bear in mind a few important facts and points.

In the first place it is as important, perhaps even more important, to know when not to attempt a breech extraction as it is to know when to perform one. This problem can best be solved after a good deal of training and adequate personal experience.

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Its solution requires fine obstetrical judgment. In my opinion a woman in labor whose infant presents by the breech is entitled to the services of an experienced obstetrician. Breech extraction should not be attempted in cases where there is evidence of marked disproportion. This is especially true in the case of the nullipara. In these instances both the mothers and babies are better served by cesarean section. This does not mean that cesarean section is the method of choice in treating breech positions; indeed, I have performed cesarean section only three times for the delivery of the breech and I have delivered vaginally some infants weighing over 9 pounds in nulliparous women.

It cannot be too strongly emphasized that no attempts at delivery should be attempted until the cervix is fully dilated. This rule should not be violated except in cases where the child is in jeopardy and even then nothing should be attempted unless the cervix is soft and fully dilatable. It is far safer to permit the fetus to perish than to attempt breech extraction through an undilated cervix. Violation of this fundamental rule ends in tragedy to either the mother or the child and oftentimes to both.

However, I believe that a goodly number of babies are lost by procrastination and a do-nothing policy after full dilatation of the cervix has been attained. This is especially true in cases of frank breech presentation. The danger of fetal asphyxiation due to interference with the circulation through the umbilical vessels is very great at this particular stage of a breech labor. My policy after the stage of full cervical dilation has been reached is not to interfere until the buttocks present at the vulva, provided there is no impairment of the fetal heart tones.

In the face of either failure of advancement or abnormal changes in the fetal heart sounds the breech is broken up and an extraction performed. It is preferable to bring down both legs but when this proves impossible the anterior leg is pulled down and its mate also as soon as it can be grasped. My technique in bringing down the leg is to go directly to the foot, grasp the child's ankle and pull it downwards and forwards. The infant's knee flexes during this motion. From here on Potter's technique is followed closely. Haste is avoided. When the umbilical cord comes into view it is pulled out a few inches on the vulva so as to avoid stretching the umbilical vessels and to minimize pressure.

In the occasional case where both shoulders cannot be delivered anteriorly, i.e., under the symphysis, I am satisfied to deliver one posteriorly.

In my opinion one of the greatest factors tending to decrease the fetal mortality in breech cases is the popularization of the "aftercoming head" forceps, for which we owe a great debt of gratitude to the late E. W. Piper. In the better obstetrical clinics one now sees less and less of the pull, agonizing to both the baby and the operator.

If a slowly performed Smellie-Veit maneuver with moderate pressure on the head by an assistant from above, does not effect prompt delivery the Piper aftercoming head forceps are immediately applied. In a few instances these forceps have slipped and I have used standard Elliott forceps and effected delivery without any trouble. I have never yet failed with the Elliott forceps.

SUMMARY AND CONCLUSIONS

One hundred three patients were delivered by breech extraction with no maternal mortality.

The gross fetal mortality was 7.76 per cent and the corrected mortality 3.8 per cent. Had these patients been under the supervision of competently trained obstetricians the corrected fetal mortality in this series might well have been in the neighborhood of 1 per cent.

Cases where appreciable disproportion exists between the passages and the passenger had best be treated by cesarean section.

Anesthesia is essential in practically all cases. I have found it rather hazardous to rely on the patient's coöperation during the course of a breech delivery. At the crucial moment she is likely to become frightened, hysterical and unreasoning and the infant can very easily be lost at this late stage. I have therefore made it a rule to anesthetize all my breech cases and thus eliminate a doubtful source of coöperation.

No attempts at delivery should be attempted until the cervix is fully dilated.

After full dilatation of the cervix delay in progress or change for the worse in the fetal heart or rhythm should call for prompt intervention.

The legs can conveniently be brought down by grasping the ankles, making traction downwards and forwards and thus flexing the knees.

At the least sign of difficulty in the delivery of the aftercoming head, forceps should be immediately applied.

BREECH positions carry a slightly greater risk to the infant, but in nine cases out of ten, the outcome is entirely satisfactory.
From—"Expectant Motherhood" by Eastman (Little, Brown and Company).

RUPTURE OF THE PREGNANT UTERUS*

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RUPTURE of the pregnant uterus is an infrequent complication of the gravid state, but the maternal and fetal mortality associated with it is so high that serious consideration may well be given to it.

In 25,935 deliveries at the Methodist Episcopal Hospital in Brooklyn between May 1, 1924 and January 1, 1939 there were fifteen cases of rupture of the pregnant uterus. The difficulty in diagnosis in such cases is indicated by the fact that three were recognized only at autopsy and it may be that others occurred without our knowledge. Seven maternal deaths and seven stillbirths occurred among the fifteen cases.

Rupture of the uterus may occur either in a uterus which has never previously suffered laceration of its musculature, or in a uterus which has been previously incised. The cases presented here were accordingly divided into primary and secondary groups. There were seven cases in the primary group and eight in the secondary. All of the secondary cases followed cesarean section. Because of the differences in etiology, diagnosis and treatment these groups will be considered separately.

In the primary ruptures six showed a laceration completely through the uterine wall, one exhibited two lacerations of the wall—neither of which extended completely through the musculature. In four cases trauma of delivery or attempted delivery was the etiologic factor in the rupture; in one case there was some question as to the part played by delivery; in one case no history or evidence of trauma could be ascertained; while in the seventh case rupture occurred apparently near the end of a short but very severe labor, in a patient who had had a previous amputation of the cervix. This last mentioned case was the one with the incomplete lacerations and was the only one in whom external hemorrhage was a factor.

TABLE 1

RUPTURE OF PREGNANT UTERUS

Total deliveries May 1, 1924, to January 1, 1939	25,935
Number of ruptured uteri	15
Maternal deaths	7
Fetal deaths	7

One of these cases occurred on the private service and six on the ward service. Four of the latter group were brought to the hospital by ambulance and had had little or no prenatal care. The one private patient was delivered by very easy breech extraction. She died within an hour of delivery and autopsy revealed a rupture along the border of a fibroid tumor. Review of this case history indicates that rupture occurred before delivery.

Two of the six ward patients had been faithful visitors to the clinic. One of these was the patient with the incomplete rupture. Evidence of hemorrhage was noted after full dilatation was reached, and midforceps were applied for delivery. This patient had had a previous amputation of the cervix and had a rather short labor characterized by extremely strong uterine contractions. In view of the evidence of hemorrhage before delivery, blame for the rupture was not placed on the forceps application. This patient bled to death and autopsy showed two incomplete lacerations high in the lower uterine segment. The other clinic case was a placenta previa treated by bag induction and delivered by

* From the Department of Obstetrics and Gynecology of the Methodist Hospital, Brooklyn, New York. Read before the section on Obstetrics and Gynecology, New York Academy of Medicine, January 24, 1939.

version—the baby weighed only 1,148 Gm. (2 pounds 8½ ounces)—and the rupture probably occurred in manual removal of the placenta. Prompt hysterectomy preceded and followed by massive transfusions gave a good maternal result.

TABLE II
TYPES OF RUPTURE

Primary ruptures	7
Maternal deaths	5
Fetal deaths	5
Secondary ruptures	8
Maternal deaths	2
Fetal deaths	2

The four ambulance cases were neglected patients who had been in labor for from twenty to forty hours with membranes ruptured for periods of seventeen to forty-nine hours. One had a transverse presentation with moderate vaginal bleeding. Bag induction was done, followed by version and extraction. The extraction was accomplished before complete dilatation and despite the size of the baby—1,956 Gm. (4 pound 5 ounces)—a laceration of cervix and uterus occurred that caused death. One patient was admitted in moderate shock. No history was obtainable because of language difficulty. The patient was treated for shock and at full dilatation was delivered of a breech presentation by embryotomy. She sustained a cervical laceration that extended into the lower uterine segment, and died. Another patient was admitted in moderate shock after instrumentation at home. She was treated for shock and delivered at full dilatation by forceps and craniotomy. Death occurred one hour after delivery and a complete laceration 6 cm. in length was found beneath the bladder. The seventh patient was fully dilated on admission with the face presenting. She was delivered without difficulty by mid-forceps after conversion of the face presentation. An attempt had been made to deliver the face as such and rupture of the uterus was noted after this attempt. Prompt hysterectomy, preceded and followed by transfusion gave a good maternal result.

Certain factors were sufficiently common to most cases in this group to attract attention. All the patients were multiparae, having had from one to nine previous pregnancies. All were in or past their middle thirties and three were 43, 44, and 45 years old, respectively. Five of the patients were definitely obese, each weighing 200 pounds or more. Contraction of the maternal pelvis was not a factor, all but one patient having normal measurements. Nor could excessive fetal size be considered a uniform cause, since there were but two oversize babies in the series—one of 10 pounds 4½ ounces and one of 10 pounds 12½ ounces. The other fetal weights ranged from 2 pounds 8½ ounces to 8 pounds 14 ounces. Abnormal presentations, however, were frequent, there being two breech presentations, one transverse and one face presentation. Placenta previa was present in two instances and bag induction was the elected method of procedure in each. In neither instance was the bag responsible for the rupture. Manual removal of the placenta and delivery through an incompletely dilated cervix were the traumatic factors in these two cases.

Shock was the most prominent symptom of primary rupture. This was not always immediate and severe, but became much more pronounced after delivery. When the rupture had occurred prior to admission to the hospital the patient was moderately shocked on admission but responded to appropriate therapy, only to suffer greater shock and death following delivery. External hemorrhage was a factor in only one case—that of the incomplete laceration. In the remainder there was no unusual vaginal bleeding. When death occurred it took place within two hours of delivery in all but one instance, one patient surviving for almost twenty-four hours. The two patients who survived were treated by immediate hysterectomy, preceded and followed by transfusions of 500 c.c. or more. One patient received 1500 c.c. of blood within ninety minutes and 500 c.c. more within the next forty-five minutes.

The most important factor in these cases is early diagnosis. This diagnosis in many

instances can be made only by inserting the examining hand inside the uterus. This procedure is warranted in any patient who comes into the hospital with a history of unsuccessful attempts at delivery at home or who is subjected to a potentially traumatic delivery in the hospital. The presence or absence of fetal heart tones is not of any consistent significance, for rupture of the uterus can occur without loss of fetal life. Abdominal palpation alone frequently gives insufficient or no evidence. In only one instance was there rupture of the anterior wall of the uterus and this rupture lay beneath the bladder. The location of the rupture and the obesity so frequently encountered caused considerable variation in the amount of abdominal tenderness elicited. A fairly consistent symptom was the cessation of labor pains after their onset, followed by steady abdominal pain. Even this was not uniformly present.

Prevention of rupture is the most important consideration and such prevention is possible. Every case of primary rupture of the uterus is a failure on the part of the physician because trauma plays so great a rôle. Delivery of the fetus—regardless of its size—before full dilatation is contraindicated. The multipara, particularly if elderly or obese, should be regarded as a potential subject for rupture of the uterus and should be managed with all the care and skill accorded to the primipara. The factors of obesity and more advanced age apparently render tissues more susceptible to the effects of traumatic delivery. It is interesting to note that while trauma was not considered a definite factor in two of our cases, there were no cases in the series where the patient had a perfectly spontaneous delivery.

Secondary rupture of the pregnant uterus presents a somewhat different situation with which to cope. Six of these patients were on the private service and two on the ward. All eight of these secondary cases followed cesarean section. They occurred in 304 repeat cesarean sections performed during this fourteen year period. In all but one instance the previous operation had been a classical one, the single exception being a previous low flap operation not performed at our hospital. The maternal and fetal mortality in the secondary group was lower than in the primary—there being only two maternal and two fetal deaths in this group. Three patients had had one previous cesarean, five had had two such operations. In all three cases where rupture followed a single previous operation, the ruptures were found in the course of routine secondary operations. No signs or symptoms of rupture were noted by the patient. These patients did well. Two were treated by suture of the rupture, and one by hysterectomy.

Of the five cases where rupture was preceded by two cesarean sections, two patients died. All five patients exhibited signs and symptoms of the rupture—sudden abdominal pain, sometimes followed by a few weak labor pains, abdominal tenderness, rigidity, increasing pulse rate and falling blood pressure. The abdominal signs were more prominent in the secondary group, undoubtedly because the rupture occurred in the anterior uterine wall. In none of these patients were the membranes ruptured nor did any of them exhibit external bleeding.

Because of the history of previous cesarean sections the diagnosis was made comparatively easily and promptly. In all but two cases operation was performed immediately after the diagnosis was made. In these two cases an unaccountable delay of three to four hours occurred. Both these patients died postoperatively and the cause of death in each instance was ascribed to pulmonary embolus. Neither diagnosis was confirmed by autopsy, but careful review of the charts tends to support the designated cause of mortality, since death was sudden and associated with unusual increases in respiratory and cardiac rates.

In one of the secondary cases the rupture occurred at seven months; in this instance the products of conception were found entirely in the abdominal cavity. This patient

was treated by suture of the ruptured scar, thus preserving the uterus, and made an uneventful recovery. No sterilization was done because of her condition but there is no record of her having become pregnant again. In the remaining seven cases, rupture occurred during the last month and in none of these was there extrusion of the uterine contents. The rupture was treated solely by suture of the wound after freshening of the edges. Sterilization was done in only two instances because of the difficulty of performing this operation in the face of adhesions and the unsatisfactory condition of the patients. Hysterectomy was done in one case, a patient who had exhibited no symptoms of rupture.

Secondary rupture of the uterus is largely a question of prevention. It occurs sufficiently frequently—eight in 304 cases—to require consideration in any patient who has been subjected to cesarean section, particularly classical cesarean section. Patients should be carefully instructed to report to the physician any unusual abdominal pain or discomfort occurring in the last trimester of pregnancy. And during this period the attention of the physician should be directed toward an evaluation of the previous scar. Any evidence of overdistention, weakness, or incipient rupture should indicate prompt operative interference.

The treatment of secondary rupture once it has occurred should, wherever possible, consist of suture of the ruptured scar. Unlike the primary ruptures, these cases require no preliminary intra-uterine exploration; nor have they usually been exposed to the dangers of prolonged labors and periods of ruptured membranes. Hence the simpler procedure of suturing the laceration is more frequently feasible. Sterilization should also be done if conditions are favorable and time permits.

Follow-up has been possible on six of the eight survivors in this series of fifteen patients. Three of those followed were potentially fertile and one became pregnant. Her pregnancy occurred within twelve months of the rupture of her uterus. Therapeutic abortion was done in the interest of this patient who had two living children and who had so recently undergone such a serious complication of pregnancy. Her convalescence was uneventful and she did not again become pregnant. The other patients followed have remained in good health and no further operative interference has been necessary in any instance.

CONCLUSIONS

1. Rupture of the pregnant uterus is an infrequent occurrence with an extremely high maternal and fetal mortality.
2. Primary rupture of the pregnant uterus is usually caused by trauma of delivery or its attempt.
3. Prevention of primary rupture depends upon constant care during labor and delivery.
4. The obese elderly multipara is particularly subject to primary rupture of the uterus.
5. Shock is the most prominent symptom of primary rupture of the uterus.
6. The treatment of primary rupture of the uterus is transfusion, hysterectomy, transfusion.
7. In our series secondary rupture of the uterus was invariably preceded by cesarean section, particularly classical cesarean section.
8. Prevention of secondary rupture of the uterus depends on the constant vigilance of the physician and the full coöperation of the patient.
9. The treatment of secondary rupture of the uterus can usually be more conservative than that of primary rupture.

CARCINOMA OF THE BREAST*

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CANCER leads to nearly 10 per cent of all deaths. It ranks second in cause of death, being surpassed only by cardiovascular disease in frequency. In cancer mortality statistics mammary malignancies rank third. In the female one-third of malignant diseases arise in the breast. Such, in brief, is the incidence and mortality of carcinoma of the breast. Barring any radical discoveries in prevention or treatment, statisticians estimate that mammary cancer incidence and mortality will continue to increase during the next thirty-five to fifty years to reach a maximum at approximately one and one-half the present rate. This increase is brought about by the greater percentage of people living into the cancer age as the result of preventive medicine, as well as a decline in the birth rate which accordingly raises the proportion of older people in the population.

It is the purpose of this paper to evaluate the results of mammary cancer treatment at the Wisconsin General Hospital from 1924 to 1936.

Like most other observers during the past quarter century we have noted no new symptoms of mammary cancer. In diagnosis we have learned that the classical signs associated with breast carcinoma in contradistinction to benign breast lesions are generally the advanced signs of this disease. Today with a better education of the laity concerning the significance of any breast tumor or symptom, an increasing number of borderline cases is brought to the physician in which a clinical diagnosis is difficult if not impossible. Therefore, we concur with the aphorism that every tumor of the breast is a cancer unless microscopic study proves it to be otherwise.

In treatment we have no new procedure but can only emphasize that the principles of "cancer cure" demand the removal or destruction of all neoplastic tissue. We believe that any mastectomy which fulfills the principles of Halstead and Willy Meyer, i.e., first, the removal of all actual and probably involved tissue within the limits of surgical access; and second, the perfection of an operative technique which interrupts efferent lymphatic channels from the breast early in the course of the operation, will, along with carefully selected irradiation therapy, produce the greater percentage of ultimate "cancer cures."

During the period of eleven years there were admitted to the hospital 318 patients for treatment of mammary carcinoma. These cases have been divided into three groups.

Group I. Patients who received primary treatment at this hospital for mammary carcinoma.

Group II. Patients who received only irradiation or no treatment at this hospital.

Group III. Patients who had received primary treatment elsewhere and were admitted here for further treatment.

In this analysis we have chosen a clinical classification similar to that of Pfahler,[1] but one which is somewhat more complex. This is not based in any sense on the microscopic grading used by Broders.

Stage 1 includes a small group of very early carcinomas 2 cm. or less in size with *no* metastases.

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Stage II designates masses larger than those of stage I but showing again no metastases. This group is further subdivided into IIa to designate that relatively rare clinical picture of an ulcerating tumor without metastases.

Stage III represents by far most frequently seen type at the surgical clinic, i.e., one with a definite carcinoma with axillary metastases. Stage IIIa includes those patients with axillary metastases and ulceration over the main tumor mass. We do not consider the latter usually as an inoperable group either with or without preoperative irradiation.

Stage IV includes the remainder of all cases permanently inoperable or upon whom a simple mastectomy is performed to rid the patient of a sloughing mass. Stage IV represents those patients with disseminated metastases (supraclavicular, en cuirasse, pulmonary, or osseous). It is further subdivided into Stage IVa which presents distant metastases plus ulceration.

TABLE I
AGE INCIDENCE OF CARCINOMA IN 177 CASES

Age Group	No. of Cases	Per Cent
20–24	0	0
25–29	2	1.2
30–34	6	3.2
35–39	16	9.1
40–44	31	17.2
45–49	21	11.8
50–54	20	11.3
55–59	27	15.3
60–64	21	11.8
65–69	21	11.8
70 and over	11	6.1
Unknown	1	1.2
	177	100.0

Group I includes 177 cases of carcinoma in which patients came for primary treatment and finally went on to an operative procedure, either radical or simple mastectomy. Seventy (39.5 per cent) of the patients were premenopausal and ninety-six (54.3 per cent) were in their postclimateric period; the menopausal status of eleven (6.2 per cent) was unknown. The tumor was primarily on the left side in ninety-four cases (53.3 per cent), on the right side in seventy-five (42.3 per cent), and bilateral in eight (4.4 per cent).

The age incidence (Table I) shows a quite steady occurrence in the age groups from 40 to 70 years. Separate, slightly higher peaks may be noted in the years 40 to 44, and 55 to 59.

Mastectomies, as previously stated, were either simple or radical. The radical type of operation, fashioned on the principles set forth in the Willy Meyer and Halstead techniques, was accomplished in 163 cases (92.2 per cent). Simple mastectomy was thought to be indicated in only fourteen cases (7.8 per cent). We feel that the indications for the latter procedure are few but definite: (1) severe cardiac disease and distinct inability to withstand a long procedure (two cases); (2) patients in stages I or II necessarily having small tumors, unwilling to withstand a radical procedure but willing to proceed with a full course of *x*-ray therapy (two cases); (3) patients over 70 years of age whose general status prohibits severe operative intervention (eight cases); (4) the presence of an ulcerating mass along with systemic metastases —in such an individual it is felt that any amputation only lessens the cares of the remaining only too short life of the patient (two cases).

In all cases, before operative procedure was undertaken, there was at least a minimum of roentgenologic investigation which usually included a flat film of the chest, anteroposterior and lateral films of the spine, and a flat anteroposterior view of the pelvis. The *x*-ray, clinical and operative findings classified the cases into their various stages. (Table II.) A paucity of stage I and IIa patients is very obvious; the former because of the rarity of such early discovery and the latter because it is rare even in a much larger series. The relationship between stages III and II is 3 to 1 and is distinctly too great to hope for monumental strides in the treatment of breast cancer.

Table II
CLASSIFICATION INTO STAGES

Stage	No. of Cases	Per Cent
I	1	0.567
II	39	22.1
IIa	1	0.567
III	116	65.5
IIIa	14	7.6
IV	5	2.9
IVa	1	0.567
	177	100.000

Postoperative mortality in the series was 3.9 per cent (seven cases). There were three chief causes of the failure of the procedure: (1) cardiac failure—three cases; (2) bronchopneumonia—two cases; (3) pulmonary embolism—two cases.

Eleven more patients (6.2 per cent) died of causes other than the carcinoma. Their span of life varied from two months to twelve years after their primary surgery. The causes of death and duration of life are indicated in Table III. This group has

Table III

Clinical Stage	Cause of Death	Period Post-operative
II	Pulmonary tuberculosis	3 years
II	Cardiac disease	6 years
II	Erysipelas	2½ years
II	Bronchopneumonia	12 years
III	Cerebral accident	4½ years
III	Carcinoma of the uterus	6 years
III	Cerebral accident	8 months
III	Pulmonary tuberculosis	1½ years
IIIa	Chronic nephritis	1 year
IIIa	Cardiovascular accident	2 months
IIIa	Bronchopneumonia	2 months

not been included in any of our final averages and we do not feel it should be. Most of these did not come to post-mortem examination here and the given causes of death in some may be questioned. In any event, only three lived beyond the five-year period and a single patient longer than ten years.

Exclusive of untraced patients (nineteen), postoperative deaths (seven), and deaths due to causes other than carcinoma of the breast (eleven), there were seventy-nine patients traced and known to be dead of carcinoma of the breast as of July 1, 1938. To this number, in all probability, might well be added the general rule of those untraced. The average duration of life varied directly with the stage of the disease at the time of surgery. (Table IV.)

Table IV
AVERAGE DURATION OF LIFE IN PATIENTS KNOWN TO BE DEAD OF CARCINOMA OF THE BREAST

Stage	No. of Cases	Average Duration of Life, Years
I	0	0
II	8	3.56
IIa	0	0
III	61	2.43
IIIa	7	.85
IV	2	.37
IVa	1	1.00

Besides the dead and untraced, there were seven (3.9 per cent) cases which had questionable or known metastases or other carcinoma when last seen here just prior to July 1, 1938:

Stage II. Two patients with questionable metastases.
Stage III. Two patients with questionable metastases.
Stage III. Two patients with known metastases.
Stage III. One patient with carcinoma of the uterus.

This leaves, as of July 1, 1938, fifty-four patients (30.4 per cent) still living and well with no evidence of metastatic lesions. Other workers have found a similar percentage of cures.[2,3,4] The postoperative period ranges from three to fourteen years and all patients are in stages I to IIIa. They are grouped in Tables V and VI.

Table V

Stage	No. of Cases	Per Cent
I	1	1.8
II	30	55.5
IIa	1	1.8
III	18	33.3
IIIa	4	7.6
	54	100.0

TABLE VI
GROUPED IN HALF YEAR PERIODS

Years Postoperative	No. of Cases	Percentage Living
3–3½	14	25.9
3½–4	2	3.7
4–4½	7	12.9
4½–5	4	7.6
5–5½	6	11.2
5½–6	0	
6–6½	2	3.7
6½–7	2	3.7
7–7½	1	1.8
7½–8	0	
8–8½	3	5.7
8½–9	1	1.8
9–9½	2	3.8
9½–10	1	1.8
Over 10	9	16.8
	54	100.0

Only four patients in stage II died as the result of their carcinoma, while in stage III all but eighteen were dead following their primary lesion at the time of the writing of this paper. This leaves a gross mortality rate of 13.9 per cent in stage II and 84.6 per cent in stage III.

Group II. This group of fifty-two patients received irradiation or no therapy at all. In twenty-eight (54 per cent) the neoplasm was primary in the right breast, in nineteen (36 per cent) in the left, and in five (10 per cent) it was bilateral on admission. Eleven of this group had a normal menstrual history while forty had passed their menopause. In one the menstrual status was not known. The age distribution is given in Table VII.

TABLE VII

Age	Number
40 to 44	6
45 to 49	5
50 to 54	5
55 to 59	6
60 to 64	10
65 to 69	8
70 or over	10
Unknown	2
	52

Fifteen (29 per cent) of the group received no treatment. Twelve of these patients are dead; the course in three is unknown. All except two of these were in stage IV in whom irradiation therapy for curative purposes was not indicated and for palliative purposes not necessary. The remaining thirty-seven (71 per cent) were given irradiation therapy for palliative purposes: (1) to heal ulcerating lesions; (2) to give relief of pain; (3) to check the growth of the neoplasm and possibly to prolong life. The duration of life is indicated in Table VIII. Eleven could not be traced. Of

TABLE VIII
DURATION OF LIFE FOLLOWING IRRADIATION THERAPY

Duration	Group III	Group IV
6 months	1	10
1 year	1	7
1½ years	1	
2 years	2	
3 years	3	
4 years	..	1

the remaining twenty-six all have died except one—now alive two years after the beginning of *x*-ray therapy.

Group III. Eighty-nine patients were seen at this hospital after having received previous treatment elsewhere. Their ages are shown in Table IX. In forty-five (50 per

TABLE IX
AGE DISTRIBUTION

Age	Number
30 to 34	6
35 to 39	8
40 to 44	9
45 to 49	18
50 to 54	14
55 to 59	11
60 to 64	9
65 to 69	9
70 and over	5
	89

cent) the malignancy was primarily in the right breast, in thirty-seven (41 per cent) in the left, in three (4 per cent) bilateral, and in four (5 per cent) unknown. Thirty-one had normal menses, forty-eight had passed their menopause and in ten the menstrual status was not known. Eighty-two (92 per cent) of these had had a mastectomy else-

where, with or without *x*-ray therapy. Two had received *x*-ray therapy only. Four had had a local excision and in one the type of previous treatment was not known. Recurrences and metastases occurred at various sites. (Table x.)

TABLE X
SITE OF METASTASES

Site	Per Cent
In operative scar	53–59
Ulceration	9
Corresponding axilla	15–16
Corresponding supraclavicular nodes	11–12
Other breast	7– 7
Systemic	48–54
Mainly in pulmonary tissue	11
Mainly in bones	14
Unknown	3

Treatment undertaken at this hospital consisted of irradiation therapy for fifty-one (57.3 per cent) of this group, radical mastectomy with irradiation therapy for seven (7.8 per cent), local excision followed by irradiation therapy for two (2.4 per cent), and no treatment for twenty-nine (32.5 per cent).

Of this group four patients are at present alive but with evidence of systemic metastases and three additional patients are alive without evidence of metastasis. One of these, a 57 year old white female, seen here in January, 1935, had a radical left mastectomy done elsewhere in November, 1934. At time of admission there was one small nodule over the sternum and fixed to it. She was given four series of *x*-ray treatments, the last one in March, 1936, and has remained well since, with almost complete disappearance of the presternal nodule. The second, a 63 year old white female, had a simple right mastectomy done elsewhere in June, 1932. On her admission in November, 1933, a mass was present in the right axilla and a radical mastectomy was done followed by irradiation therapy (five series). The patient at present is free of metastases. The third, a 67 year old white female, had an ulcerated mass in the right axilla following partial excision of a tumor two years previously. A biopsy in November, 1934 proved the tumor to be an adenocarcinoma of the lingula of the right breast. Six series of *x*-ray treatments were given and the patient is at present without demonstrable metastases. However, an indefinite indurated mass does remain in the right axilla which on biopsy shows a chronic inflammatory reaction.

Twelve of the patients could not be traced and three died of causes unassociated with their malignancy. All the remaining patients (sixty-eight) are known to have died primarily as the result of the mammary neoplasm. Their duration of life with type of treatment is given in Table xi.

TABLE XI
DURATION OF LIFE AFTER TREATMENT

Life Span	None	Treatment *X*-ray	Mastectomy	Local Excision with *X*-ray
6 months	7	17	1	1
6 mo. to 1 yr	3	4	..	2
1 to 1½ yr	2	7		
1½ yr. to 2 yr	5	5	1	
2 to 2½ yr				
2½ to 3 yr	..	2		
3 to 3½ yr	1	3		
3½ to 4 yr	..	2		
4 to 4½ yr	1	1		
4½ to 5 yr	..	1		
5 to 5½ yr	1			
5½ to 6 yr	..	..	1	
Total of 68 cases				

CONCLUSIONS

We feel that *x*-ray irradiation has become an important adjunct in the treatment of carcinoma of the breast. Our experience in preoperative treatment has been limited to a few cases during the last year and one in the preceding ten year period covered in this analysis. Stage III, we believe, is the most important group in which irradiation before surgery is given although results are, as yet, too recent to publish in this paper. Ablation of the ovaries in premenopausal patients, especially those individuals in their third and fourth decades with tumors in Stages I, II, and III, is now based on long standing biological facts.[2] Where it is possi-

ble we suggest that it be a temporary procedure but the uncertainty of this *x*-ray dosage often makes it permanent. In this end result by irradiation our purpose has been defeated by two important factors which are worthy of consideration: (1) maternal instinct and sincere desire for continued fertility and ability to procreate; (2) religious obstruction.

During the period 1925 to 1929 inclusive (fifty-two cases) only eighteen received postoperative irradiation and a single case had preoperative *x*-ray treatment. In the rest of the series 1930 to 1935 (125 cases) all but four patients received a postoperative series. Of the four only one had a distinct contraindication, i.e., pulmonary tuberculosis. All patients with resectable lesions are now receiving *x*-ray after surgery. Several times we have noted recurrences five to ten years after operation in patients who had small lesions and received no *x*-ray therapy.

Stage IV and IVa patients, unless they are hopelessly riddled with metastatic lesions, are thought to be worthy of irradiation. One particular patient in the past two years entered the hospital as a stage IVa with bilateral pleural effusion. The pleural fluid revealed cancer cells. Besides this she had almost a complete collapse of D_{12}. She was given a series of treatments, had several thoracenteses and was fitted with a Taylor back brace. Three months later a simple mastectomy was done and the spine was irradiated. By religious *x*-ray treatment her life was prolonged two years and without the suffering often used as an excuse for not treating late cases.

An outline of the general principle followed in the postoperative irradiation of carcinoma of the breast is somewhat as follows:

In cases where the growth is definitely limited to the breast a three field technique is used. The anterior breast area extends from the clavicle to the level of the diaphragm and from the midsternal line to the midaxillary line. The supraclavicular field is limited by the superior border of the clavicle, the medial border of the sternomastoid muscle and the superior border of the trapezius. The limitations of the axilla are well known.

The physical factors used may be summarized in this manner. The anterior breast area receives 200 r, measured in air, at 100 Kv. 5 ma. 30 cm. HVL cu. 0.14 mm. for a total dosage of 1200 r. The supraclavicular and direct axilla each receive 200 r, measured in air, at 160 Kv. 7.5 ma. 30 cm. HVL cu. 0.85 mm. for a total of 1200 r. A second series of treatments is given after three months, a third, fourth and possibly a fifth series after four months interval.

When the axillary nodes are involved or suspected of involvement, a modification of the above procedure is followed. Instead of irradiating the axilla through one portal, two portals are used, one through the anterior axillary fold and the other through the posterior axillary fold, with the same physical factors as in the one portal technique. In addition, the total dosage of the initial series is increased to 1600 r to all areas, but in subsequent series, a total of 1200 r is given.

This plan is not hard and fast, but is variable according to the demands of each individual case.

SUMMARY

In the differential clinical diagnosis of mammary neoplasms, adenocarcinoma can safely be excluded only after biopsy and microscopic study.

In a series of 177 cases of mammary cancer treated by surgery with *x*-ray therapy, 30.4 per cent of the patients are alive three years or more after treatment; 18 per cent five years or longer.

Only one of every four patients presents herself with a lesion still within the breast. Exclusive of Group I this results in a cure in 86.1 per cent in Group II and 15.4 per cent in Group III. The mortality rate then increases five and one-half times as spread occurs beyond the breast.

The results of irradiation therapy for *curative* purposes in recurrent or metastatic mammary adenocarcinoma are poor.

PERCENTAGE OF CURE BY STAGES

Stage	Patients Treated	Per Cent Cures
I	1	100.0
II and IIa	40	86.1
III and IIIa	130	15.4

The indications for simple mastectomy in carcinoma of the breast irrespective of the stage of the tumor have been presented.

If for no other indication than to give the patient the benefit of all possible curative therapy, we shall continue to use irradiation therapy with surgery in the treatment of all operable mammary carcinomas.

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Usually biopsy should not be performed unless the surgeon has facilities to have the tissue examined by the pathologist grossly or microscopically while the patient is on the operating table and is prepared to proceed immediately with the radical operation if the tissue examined reveals malignancy.

From—"Minor Surgery" by Christopher (Saunders).

SURGICAL TREATMENT OF GASTROINTESTINAL HEMORRHAGE

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THE surgical treatment of gastrointestinal hemorrhage is frequently a debatable and difficult problem from the standpoint of successful management. We propose to mention a few of these problems and to consider briefly some of the surgical procedures employed in their treatment. The conditions that cause hemorrhage can be divided anatomically into gastroduodenal and extragastroduodenal. In more than 75 per cent of cases,[4] gastrointestinal hemorrhage is due to the gastroduodenal group of conditions, and for this reason we shall be concerned chiefly with this group.

Of primary importance, before one contemplates operation for any gastroenteric hemorrhage, is an accurate diagnosis; for, with the exception of a few conditions, such as purpura hemorrhagica, mesenteric thrombosis, intussusception, volvulus and certain cases of peptic ulcer, the immediate remedies that should be employed are entirely medical. If doubt exists as to the diagnosis, it is usually the best policy to treat the patient conservatively.

PEPTIC ULCER

By far the most common source of hemorrhage in the gastroduodenal group is a peptic ulcer. It is generally agreed that 12 to 20 per cent of patients who have peptic ulcer have gross hemorrhage and the reported mortality in their treatment by conservative means varies from 1 to 25 per cent.[3] Some authors advocate the immediate surgical treatment of all bleeding peptic ulcers. Finsterer has stated that operation during the first forty-eight hours carries a mortality of less than 5 per cent, but that after forty-eight hours this increases to 30 per cent. Others feel that when adequate conservative treatment is employed the mortality will be less than 5 per cent and therefore immediate operation is only rarely justified.

Indications for Surgical Intervention. There are a few cases, however, in which immediate operation should be employed, but the selection of these is difficult. All patients who have gastrointestinal hemorrhage due to peptic ulcer should be seen immediately by both surgeon and internist, and certain factors should be considered; namely, what is the patient's general condition, is he in a state of collapse and exsanguinated? Has he had a severe massive hemorrhage or is the bleeding slowly and progressively going on? One helpful point in this regard is to note whether or not the vomitus is bright red and alkaline, or coffee-ground and acid. The former is indicative of a profuse and rapid process and the latter of a comparatively slow one. The patient's age is of importance, as Goldman has estimated that patients more than 40 years old are more than 70 per cent more likely to die than those under 40. Allen's series showed a mortality of 33 per cent for patients more than fifty years of age, as compared with a mortality of 4.4 per cent for patients less than fifty years of age.

The presence of arteriosclerosis is an important factor. In various series, approximately 70 per cent[7] of those patients who have died have shown gross arteriosclerosis. Women are less likely to have peptic ulcer than men, but those women who do are equally prone to bleed profusely. Women seem also more likely to have spontaneous remissions. Among patients whose concentration of hemoglobin is less than 60 per

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cent[8] the mortality rate is four times greater than that among those whose concentration of hemoglobin is greater than 60 per cent (Dare). In general, two or more gross hemorrhages may mean that further recurrences are in store and after the second hemorrhage an abrupt rise in the mortality rate occurs.

Assuming that an operation be performed, what is the possibility of arresting the hemorrhage with certainty? Is the peptic ulcer in the stomach or duodenum? Is it acute or chronic? The presence of the hemorrhage itself adds greatly to the technical difficulties and may make it impossible to locate the lesion. Prior to operation it is an arbitrary point as to whether a gastric or duodenal ulcer is present. If a gastric ulcer is present, it may be irremovable because of its situation. Even if the ulcer can be identified, its situation and surroundings may not allow efficient ligation of the bleeding points. If the source of bleeding is due to an acute ulcer, gastritis, or duodenitis, the source may be identified only after opening the stomach and duodenum.

When these factors have been considered, even though the patient has a peptic ulcer and other possible causes for hemorrhage have not been found, the question of operation is still difficult. It is likely that, if the patient is less than 50 years of age and has elastic vessels, conservative measures are, temporarily at least, indicated. If, however, the patient is more than 50 years of age, has an apparent degree of arteriosclerosis and at the same time is a fair surgical risk, operation must be seriously considered. If under conservative treatment the bleeding continues, and if the hemoglobin cannot be controlled by multiple transfusions, a surgical attack on the ulcer is sound treatment. In other cases, in which the ulcer has caused an erosion of some large vessel or a concomitant hemorrhage and perforation, operation is imperative. Hemorrhage in a case in which there is recent roentgenographic evidence of an ulcer on the posterior wall of the duodenum, or pyloric or duodenal stenosis, frequently justifies operation. Surgical treatment must be considered when a second hemorrhage occurs soon after the first, or when the patient relates a long, definite history of ulcer and has a fibrotic ulcer.

Surgical Treatment. The surgical procedure employed will vary with the type and site of the ulcer, the condition of the patient and the skill of the particular operator. If a gastric ulcer is found, either gastric resection or some form of an excision with a gastrojejunostomy may be done. If the operation is one of emergency, however, only ligation of the bleeding vessels may be justified. These vessels must be ligated in healthy tissue, outside the wall of the stomach. They can best be ligated by opening the normal anterior wall of the stomach, so that the region from which bleeding occurs can be visualized and compressed during ligation of the anastomosing branches. When this is accomplished, the opening in the wall of the stomach is closed and a resection is carried out at a later date.

If the ulcer is duodenal, several methods of treatment may be employed. Occasionally, the duodenal ulcer may be excised and a gastroenterostomy performed. This procedure is most desirable for patients more than 50 years of age, and for those who have an ulcer on the anterior wall. If the ulcer is on the posterior wall of the duodenum, as is usually the case, several methods must be considered. The radical operation of gastroduodenal resection with excision of the ulcer is to be preferred if there is little inflammatory reaction and if the patient's condition permits. More frequently than not, considerable induration and inflammation may make a radical gastroduodenal resection impossible. A gastric resection down to the pylorus, thus excluding the ulcer, is feasible in these cases. One can neither open the anterior wall of the duodenum and successfully place ligatures in the friable bed of the ulcer nor safely pass stitch ligatures in such a way as to occlude the vessels in healthy tissue from within.

The superior pancreaticoduodenal artery is usually the artery involved in the hemorrhage, but due to the diffuse arterial anastomoses behind the duodenum, it is unlikely that bleeding can be controlled except by ligation of the right gastroepiploic artery and the superior and inferior pancreaticoduodenal arteries. Allen and Benedict transect the prepyloric end of the stomach, elevate the duodenal end and separately ligate the blood vessels outside the duodenum before the ulcer is approached. Others propose an under-running and ligation of the blood vessels in the region of the pylorus and of the first part of the duodenum. Some surgeons open the pylorus and anterior wall of the duodenum for exposure and then cauterize or otherwise obliterate the ulcer. The anterior wall of the duodenum may be infolded in such a manner as to produce stenosis and plugging of the bed of the ulcer, or the pylorus may be occluded with a heavy suture. A posterior gastroenterostomy is to be performed as an adjunct to these procedures. In other cases pyloric exclusion with a gastroenterostomy may be done, or occasionally a gastroenterostomy alone. As a rule, however, any indirect operation without a direct attack on the ulcer will fail.

When the hemorrhage ceases, or subsides under conservative treatment, the problem of surgical treatment is debatable. If the patient is young and responds well to a dietary regimen, we may justly feel that a radical operative procedure is unnecessary. If the patient is more than 50 years of age, or has had several hemorrhages, some surgical procedure usually should be carried out. It is probable that the greatest protection can be obtained from the more radical procedure, although in certain cases a simple gastroenterostomy will protect the patient against recurrent and, at least, fatal hemorrhage.

Gastrojejunal Ulcer. A few patients who have had a gastroenterostomy performed, experience an acute massive hemorrhage associated with a stomal ulcer. The inflammatory process may involve and erode a large vessel such as the gastroepiploic or the colic arch. This presents a difficult surgical problem because it is necessary to take down the old anastomosis in order to find the bleeding ulcer, and if such a procedure is to be done, it must be done early. An acute massive hemorrhage from a stomal ulcer is uncommon, although in 35 per cent of such cases,[10] the ulcers do bleed. When such a hemorrhage occurs, it is usually slow and progressive, and it is best treated conservatively until the patient's condition warrants operation.

GASTRIC CARCINOMA

Gastric carcinoma is frequently the cause of gastrointestinal bleeding, but is rarely the cause of a massive hemorrhage. If a patient is known to have a carcinoma of the stomach, and has an acute massive hemorrhage, the advisability of performing an immediate resection must be considered, as it may require weeks, or even months, to overcome the effects of this serious hemorrhage.

POSTOPERATIVE HEMORRHAGE

Postoperative hemorrhage in cases in which some surgical procedure has been performed on the stomach or duodenum, is usually best treated by conservative measures. Occasionally, however, in spite of conservative therapy, the patient's condition may necessitate reoperation. If a posterior gastroenterostomy has been performed, one of two procedures may be employed. The anterior wall of the stomach may be opened, the anastomosis may be pulled up through this opening, and if one or two single bleeding points are seen, they are controlled by sutures. However, a general oozing is more frequently present; in which case it is better to oversew the entire circumference of the gastroenterostomy. An alternative method is to remove the anterior row of sutures of the anastomosis, thus visualizing the posterior row. The bleeding is controlled and the anastomosis remade. To make other types of anastomoses, it is

usually necessary to take down a portion of the anastomosis in order to visualize the bleeding points. If, in the previous operation, the original ulcer was not dealt with and if, after examination of the line of suture, it is obvious that this is not the source of the hemorrhage, a direct attack on the bleeding ulcer must be considered.

MISCELLANEOUS SOURCES OF HEMORRHAGE IN THE GASTRODUODENAL GROUP

Gastric polyposis, gastritis, duodenitis, syphilis and certain benign tumors of the stomach have been reported in isolated instances as causes of acute massive hemorrhage. This is unusual, but when it does occur, it should probably be treated during the acute phase conservatively, and operation should be performed during the interval.

Varices of the esophagus may cause acute massive hemorrhage; approximately 5 per cent[10] of the deaths in cases of portal cirrhosis are due to hemorrhages from this source. Emergency operation cannot be recommended in these cases. When the diagnosis is in doubt, one is justified in carrying out roentgenologic examination of the esophagus. Occasionally, during splenectomy, or some other surgical procedure, it is advisable to attempt ligation of some of these varicosities, or to ligate the coronary vein. Some have injected sclerosing solutions directly into these veins, but in the majority of instances surgical treatment is not satisfactory.

MISCELLANEOUS SOURCES OF HEMORRHAGE IN THE EXTRAGASTRODUODENAL GROUP

Gastrointestinal hemorrhage is a frequent complication of certain forms of splenic disease, namely splenic anemia, or Banti's disease, and thrombocytopenic purpura. Hemorrhage is seen in the late stages of nearly every case of splenic anemia. A splenectomy should be performed as early as possible in the course of the disease, but operation should not be performed during or just after a hemorrhage, but usually during the interval when the patient is the best operative risk.

Rather dramatic results may be obtained by performing a splenectomy during the active hemorrhagic state of thrombocytopenic purpura. Pemberton said: "In the absence of an associated condition which would constitute a definite contraindication, it is my conviction that unless the course of the disease is checked by one or possibly two transfusions of blood, splenectomy should be done without further delay, for the risk of tiding these patients over the acute exacerbation is often greater than that of operation during the early phase of the crisis of the disease."

Meckel's diverticulum and solitary ulcers of the ileum may occasionally be the source of rather puzzling gastrointestinal hemorrhage. It is estimated that 2 per cent of the entire population have a Meckel's diverticulum, but only one-fourth of these show some pathologic change.[5] The diagnosis of either of these conditions can be made only by exclusion. It is important to remember that this condition exists and to look for it on abdominal exploration.

Three conditions that constitute emergencies should be mentioned briefly. They are mesenteric embolism and thrombosis, volvulus, and intussusception. Intussusception is rare in adults, but should always be suspected in infants when fresh blood is passed from the rectum. The same may be said to apply to volvulus in adults. A vascular accident to the mesenteric vessels may cause rapid effusion of a large amount of blood into the bowel. Embolism of the artery and thrombosis of the veins are rare, but the former should be suspected in cases of advanced rheumatic heart disease or subacute bacterial endocarditis. If the diagnosis is made early, a resection may be performed.

A difficult problem is presented when a patient has recurring hemorrhages, the cause of which cannot be established definitely because of the absence of other symptoms, and because of negative or inconclusive findings on roentgenologic study

of the gastrointestinal tract. Exploration should be advised in such cases. A lesion of the duodenum frequently is discovered, usually on the posterior wall and farther from the pylorus than such lesions commonly occur. If the stomach and duodenum appear normal, and further exploration fails to reveal a satisfactory cause for the bleeding, one is justified in opening the stomach or duodenum so that a more accurate examination can be made. The other abdominal viscera such as the gall-bladder and appendix should be examined carefully to make certain that some other pathologic lesion that could account for the bleeding is not present. If any such condition is present, the surgeon should not hesitate to deal with it radically.

The majority of the common causes of gastrointestinal bleeding have been mentioned. Many other conditions, such as lesions in the large intestine, rupture of an aneurysm, acute toxic states with erosion of the stomach, congestive heart failure, duodenal carcinoma, syphilis, vicarious menstruation, tuberculosis, primary blood dyscrasias, trauma, vitamin deficiency, hemorrhagic diathesis and so forth, could be considered. The surgical treatment of gastrointestinal hemorrhage is usually indicated by the diagnosis.

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AN OPERATION FOR THE REPAIR OF DIASTASIS RECTI ABDOMINIS*

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THE various methods employed at present in the repair of diastasis of the recti abdominis muscles give a fairly high percentage of successful results, but are followed by recurrence of the original weakness in a certain number of cases. The search for new methods of repair therefore still continues.

The purpose of this communication is to describe a technique which I have used with increasing satisfaction for the last three years in cases in which the diastasis extends above the linea semicircularis, that is, above the inferior termination of the posterior rectus sheath. Although the attenuated anterior and posterior rectus sheaths, transversalis fascia, and peritoneum above the linea semicircularis are of themselves insufficient for support of the mid-abdominal wall in such cases, in many instances these structures may be utilized to form two of the layers of an adequate three-layered closure, the third of which is muscle. (Fig. 1E.)

The use of this technique requires a preliminary consideration of the anatomy of the rectus sheath, which differs above and below the linea semicircularis. Above the linea semicircularis the rectus sheath is formed by the aponeuroses of the external oblique, internal oblique, and transversus abdominis muscles as follows:

At the lateral edge of the rectus muscle the aponeurosis of the internal oblique divides into two layers, one of which passes in front of the rectus muscle and fuses with the aponeurosis of the external oblique, while the other passes behind the rectus muscle and fuses with the aponeurosis of the transversus abdominis. The aponeuroses of all these muscles fuse at the medial border of the rectus, where they are inserted into the linea alba. The posterior sheath of the rectus muscle extends from the margins of the costal cartilages inferiorly to a level about midway between the umbilicus and the symphysis pubis. The inferior termination of the posterior rectus sheath is referred to as the linea semicircularis. Below this level the muscle lies directly upon the transversalis fascia, without a posterior sheath, and the aponeuroses of the external and internal oblique muscles and of the transversus abdominis all pass in front of it to form its anterior sheath. The thickness of the anterior sheath is therefore greater below than above the linea semicircularis.

TECHNIQUE

The operation to be described differs from other procedures devised for the repair of diastasis of the recti abdominis muscles in the method of utilization of the fascial structures. It is performed as follows:

A midline incision is made over the entire extent of the separation of the recti muscles, extending slightly beyond the diastasis at each end. The umbilicus is excised. The rectus sheaths are exposed, the dissection being carried back on the right side to a point at which the rectus muscle can be palpated. On the left side the dissection is carried only far enough back from the midline to allow sufficient room for suture of the medial edge of the sheath on that side.

The linea alba and the peritoneum are opened in the midline over the entire extent of the separation of the muscles. By careful

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inspection and palpation it is then determined whether the combined fascial layers will be sufficiently strong to serve as a single layer in the closure. If the technique seems applicable in the special case, the right anterior rectus sheath is incised to permit entry into the compartment occupied by the rectus muscle, the distance of the incision from the midline being determined by the amount of tightening or bracing of the abdominal wall that seems necessary.

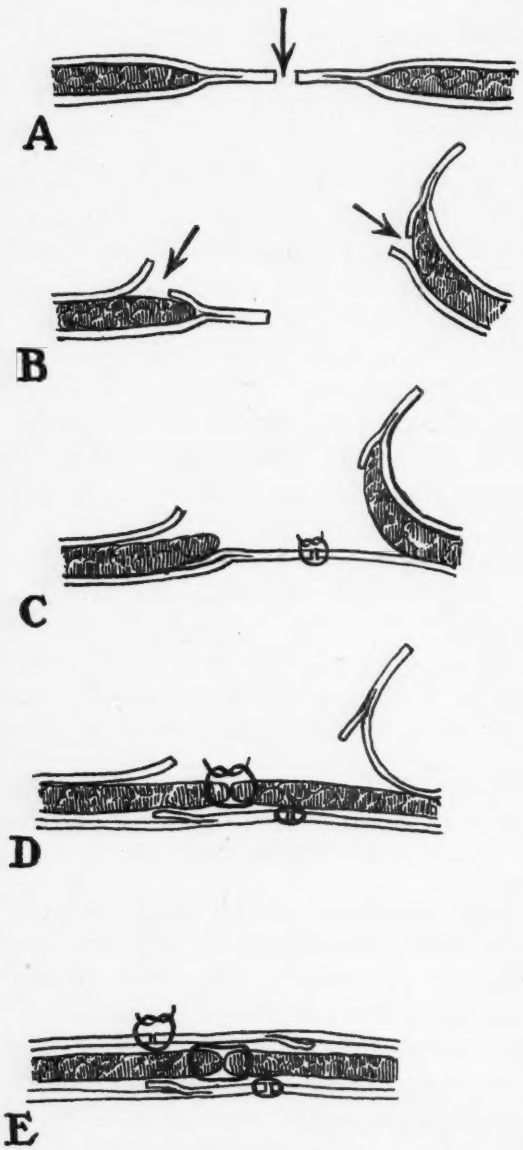

FIG. 1. Diagrammatic cross sections showing method of obtaining the flaps and their use in the closure of the diastasis. A, midline incision through the attentuated rectus sheaths, transversalis fascia, and peritoneum. B, incisions into the anterior rectus sheath on the right side and into the posterior rectus sheath on the left. C, approximation of long and short posterior flaps. D, approximation of posterior flaps and rectus muscles. E, approximation of anterior flaps, muscles, and posterior flaps. Note relations of the suture lines to each other in the three layers of the closure.

The incision in the right anterior rectus sheath is then enlarged upward and downward, parallel to the midline incision, but curving toward it at the extreme upper and lower ends. If the separation of the rectus muscle extends below the linea semicircularis, the incision in the rectus sheath curves toward the midline at that level. The anterior sheath is freed from the muscle as necessary.

When the dissection is complete, a long posterior flap has been provided, consisting of a portion of the anterior rectus sheath plus the intact posterior sheath, transversalis fascia, and peritoneum. A short anterior flap is also provided, consisting of the anterior sheath of the rectus muscle. (Fig. 1B.) The long posterior flap is always made on the right side, as it is desirable to have the round ligament of the liver included in it.

The left edge of the wound is now turned outward, so as to expose the inner side of the peritoneum. An incision is made from the peritoneal side through the peritoneum, transversalis fascia, and posterior sheath of the rectus muscle into the compartment occupied by the muscle. (Fig. 2.) The incision is continued upward and downward parallel with the midline incision, as on the right side, and is curved in the same way toward the midline at either end. There have now been provided on this side a long anterior flap, consisting of a short portion of peritoneum, transversalis fascia, and posterior rectus sheath, plus the intact anterior rectus sheath, and a short posterior flap, consisting of the posterior sheath of the rectus muscle, transversalis fascia, and peritoneum. (Fig. 1B.)

If the original midline incision does not extend below the linea semicircularis, the two flaps described are continuous above and

below with the linea alba. If it extends below the linea semicircularis, the long posterior flap is continuous inferiorly with the trans-

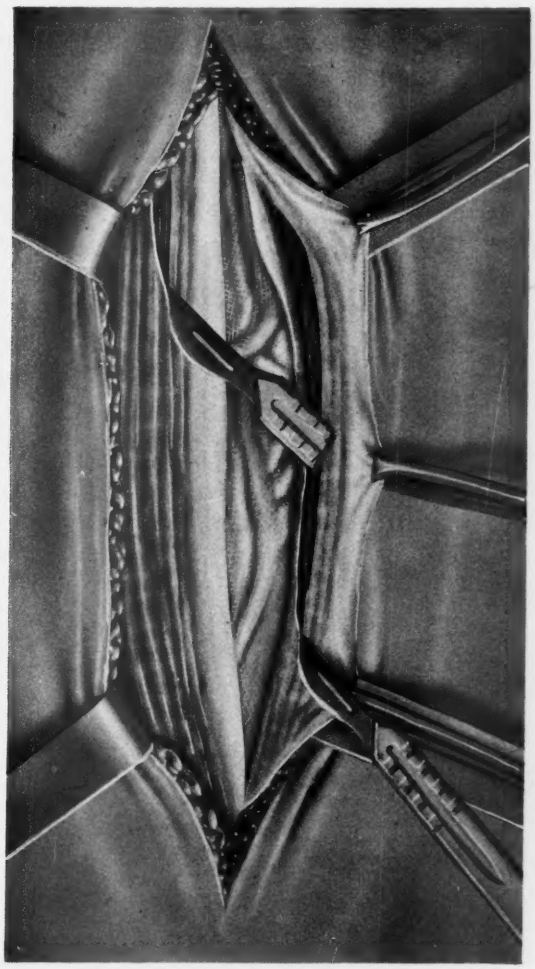

Fig. 2. Peritoneal cavity opened in the midline. Incisions are made into the right rectus sheath anteriorly and into the left rectus sheath posteriorly.

versalis fascia and peritoneum, and the long anterior flap is continuous inferiorly with the anterior rectus sheath.

In the closure of the incision, the first suture line is begun in the linea alba at the upper end of the incision. The long posterior flap is sutured to the short posterior flap (Fig. 1C) by sutures so placed as to appose the peritoneal surfaces and continued until the incision is completely closed. When the diastasis extends below the linea semicircularis, the suture line is continued below that level, but the sutures are placed only through the peritoneum and transversalis fascia.

Interrupted sutures are next placed in the muscles which have been freed during dissection of the flaps, but are not tied at

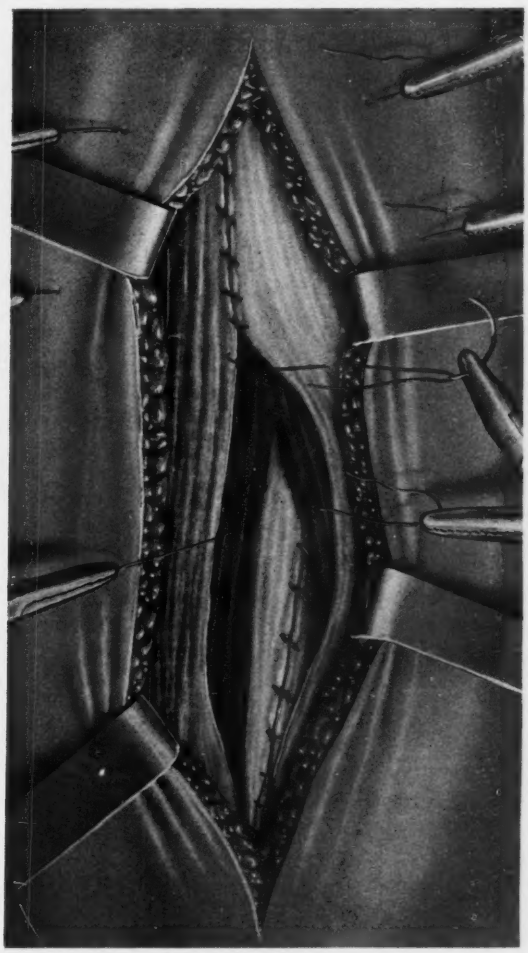

Fig. 3. Approximation of long and short posterior flaps. Interrupted muscle sutures and two retention sutures in place. Continuous suture of long and short anterior flaps has been begun. (Some of the interrupted muscle sutures and retention sutures have been omitted in the drawing to make the demonstration clearer.)

this time. (Figs. 1D and 3.) If it is thought desirable to use retention sutures, they are placed from within outward through the anterior rectus sheath and the skin.

The third line of closure is started in the linea alba at the upper angle of the incision, just beyond the point at which the first layer of sutures was started. This suture line is continued downward to unite the long anterior flap and the short anterior flap. (Figs. 1E and 3.) When the separation extends below the linea semicircularis, the suture line is continued below this level to

approximate the right and left anterior rectus sheaths, which should be overlapped sufficiently far to obtain the desired amount of tightening in the lower part of the abdomen. The interrupted sutures already placed in the muscles are tied as they are reached.

COMMENT

The technique described for the closure of diastases of the recti abdominis muscles has been used in eleven cases, which have been followed up for periods ranging from six months to three years. The results have been entirely satisfactory and there has been no evidence of recurrence in any case.

This method of repair possesses several distinct advantages. It can be used with safety as a supplementary procedure in operations undertaken primarily for other purposes, because it adds very little to the time required for simple incisions and closures of similar extent. I have personally used it in operations undertaken for surgery of the biliary tract and the pelvic organs, and have encountered no difficulties in the performance of the combined operations.

All the fascial structures are employed in the closure, which is therefore as strong as it could possibly be. An additional safeguard lies in the fact that the approximation of each fascial layer is done at a different point (Fig. 1E), so that the strain of intra-abdominal pressure is widely distributed. The technique is logical from the anatomic point of view, and permits a closure more nearly approximating normal anatomic restoration than is possible in any other procedure for this purpose which has come to my attention.

ACUTE APPENDICITIS IN DIABETES MELLITUS*

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THE diagnosis and treatment of acute appendicitis in diabetic patients is a serious problem because of the presence of a metabolic disease as an associated factor at the outset. The two conditions existing together have a bad influence on each other. While an early diagnosis of acute appendicitis is always desirable, it is particularly important in diabetic patients. In mild diabetics under treatment, the diagnosis in typical cases is no more difficult than in non-diabetics, other things being equal. In the severe cases, especially those not under treatment, the diagnosis may be more difficult and the surgical intervention rather late. Early removal of an acutely infected appendix is more urgent in a diabetic than in a normal person for two reasons: first, the infection quickly aggravates the diabetic condition; second, the infection does not remain localized long. In addition, we must consider that patients in acidosis are encountered in whom acute appendicitis is either suspected or actually present, and that acidosis itself often gives rise to diffuse abdominal pain, tenderness, and vomiting.

Recent personal experience with two cases at the Mt. Sinai Hospital, Chicago, prompted me to review the subject. The hospital records were searched for cases of acute appendicitis in diabetic material from January 1, 1929 to January 1, 1939. During this time 1,251 diabetic patients were admitted to the hospital for various ailments. Acute appendicitis occurred eight times. Seven of the eight were complicated at the time of operation by gangrene, perforation, abscess formation, or peritonitis. Two had gangrene, two had abscess formation, three had peritonitis. Of the last group, two died postoperatively. One patient had acute catarrhal appendicitis. One had acute clinical symptoms but pathologically the appendix was not acutely inflamed; this case is not considered in this paper.

With the exception of one case, the patients were in the elderly age group, the ages being, 14, 47, 49, 50, 55, 55, 60, and 63, respectively. There were six females and two male patients. Heart disease, hypertension, arteriosclerosis, and obesity were associated factors.

Bothe and Bearwood[1] reviewed 1,260 cases of diabetes mellitus admitted to the Pennsylvania Hospital. In this series of cases there were seven instances of acute appendicitis, and of these four were ruptured at the time of operation. These authors emphasize the difficulty at times of an early diagnosis and proper evaluation of abdominal symptoms in a diabetic patient with an acute surgical lesion. In their material, 136 patients of 1,260 studied had diabetic acidosis, and of these "74 per cent presented abdominal symptoms of nausea, vomiting and abdominal pain which were usually associated with fever and leucocytosis."

McKittrick and Root[2] make this terse statement on the subject in their book on diabetic surgery: "Acute appendicitis is especially dangerous in a diabetic because the symptoms and signs may be obscure, the diagnosis difficult and the treatment late." In the fourteen cases of acute appendicitis listed in their book, six were ruptured and four gangrenous, i.e., ten in fourteen were complicated at the time of operation.

McKittrick[3] reports thirty-seven operations for appendicitis in which twelve required drainage. Of these twelve six cases had drainage of abscess with or without spreading peritonitis. The mortality in the

* From the Medical and Surgical Service, Mt. Sinai Hospital, Chicago, based on a review of 1,251 diabetic patients from January 1, 1929 to January 1, 1939.

last six cases was 33.3 per cent, while the total mortality was 6.1 per cent.

TABLE I

RELATIVE FREQUENCY OF CLINICAL SYMPTOMS ON ADMISSION

Onset sudden	6
Onset insidious	2
Pain in right lower quadrant	8
Tenderness in right lower quadrant	6
Rebound tenderness in right lower quadrant	3
Muscle spasm or rigidity	5
Pain in lower half of abdomen	3
Nausea	1
Vomiting	0
Temperature	8
Leucocytosis	7
Appeared acutely ill	3
Distention of lower half of abdomen	4
Mass in right lower quadrant	2
Acidosis present	2

Signs and Symptoms. Table 1 shows that abdominal pain with localization in the right lower quadrant, local tenderness, muscle spasm or rigidity, temperature, pulse of 100 to 120, and leucocytosis form the signs and symptoms of acute appendicitis in the group reviewed. Nausea and vomiting were conspicuously absent.

The onset was sudden in six and insidious in two cases, although McKittrick and Root[2] claim that the onset is generally insidious in acute appendicitis in diabetic persons. All had an elevated temperature and pulse. All but one had leucocytosis; in this patient the white count was 9,900 and differential 79 per cent. Ruptured appendicitis with peritonitis was present. Those who had pain in the lower half of the abdomen (right more than left), distention, and temperature of 101 to 102°F., suffered from ruptured appendicitis with peritonitis. Of the two patients with masses in the right lower quadrant, in one there was spontaneous rupture into the rectum. The other was drained. Recovery followed in both cases.

In the three cases of peritonitis, the extent of the pathologic process was in a

TABLE II*

R.B.C.	W.B.C.	Neutrophiles, Per Cent	Hgb.	Urine Sugar	Blood	Temp. Rectal	Pulse	Laxative
5,100,000	17,400	82	90	2.5% A+++	147	102.2	110	Enema
4,000,000	13,100	84	72	1.6%	221	102.8	120	No mention
.....	17,300	87	?	+ A++++	181	100.2	100	Yes
4,300,000	9,900	79	?	3.8% A++	286	102.4	100	Yes
3,900,000	17,800	82	75	+ A	195	100.6	115	No mention
4,000,000	17,000	79	?	3.3% A+	251	101.2	108	Yes
.....	26,650	83	?	5% A+++ D+	371	102.4	116	Yes
.....	14,200	88	?	4% A++ D+	286	101	110	Enema

*A = Acetone
D = Diacetic acid

clinical sense underestimated. One patient, in whom there was a tendency to localize in the right lower quadrant and pelvis recovered, but had extensive wound infection with slough of fascia; the other two died shortly after operation. In two cases of appendiceal abscess there was a positive diacetic acid test. These were in acidosis.

Therefore, the diagnosis of acute appendicitis in a diabetic person may be based on a history of abdominal pain which localizes in the right lower quadrant, local tenderness and muscle spasm, termperature 100 to 101, or even 102, elevated pulse, leucocytosis. Likewise, appendiceal symptoms with pain and distention in the lower half of the abdomen should give a suspicion of peritonitis.

A differential diagnosis between acute appendicitis and acidosis with abdominal symptoms simulating a surgical lesion may be a difficult problem. In appendicitis the onset is rather sudden and pain and tenderness are localized to the right lower quadrant, unless abscess or peritonitis is a complication. In acidosis, the onset is usually insidious and pain and tenderness are likely to be diffuse. With insulin therapy, the symptoms in acidosis will clear up or become milder in three to six hours, whereas in appendicitis the symptoms remain the same or get worse as one waits. A positive diacetic acid test is characteristic for acidosis and in its absence one is fairly safe in saying that acidosis does not exist. Blood sugar and carbon dioxide combining power of blood plasma will help in difficult cases to establish proper diagnosis. Finally, if clinical symptoms point to acute appendicitis and there is no mitigation of symptoms after insulin therapy, prepare the patient for surgery.

Pre- and Postoperative Care. The decision as to the urgency of immediate operation is of vital importance. The immediate preoperative and postoperative care consisted in most instances of insulin subcutaneously based on urinary analyses for sugar, acetone, diacetic acid, and infusion of physiologic salt solution and 5 per cent glucose.

Opinions differ as to the needs of glucose immediately before and after surgery. Earlier writers on the subject, Jones, McKittrick, Sisco,[4] and Walter, Myerding, Judd, Wilder[5] favored insulin subcutaneously and salt solution preferably by the hypodermoclysis route. Woodyatt[6] is also of the opinion that glucose is not needed immediately before surgery. The reason given by him is that the patient already has enough glucose which he cannot utilize.

Standard and his associates[7] state that he gives for all major surgical procedures an infusion of 1000 c.c. of physiologic salt solution of sodium chloride with 50 Gm. of dextrose two hours before operation and repeats this postoperatively in all moderately severe cases.

Table III

Sex	Age	Postoperative Diagnosis	Operation	Anesthesia
M	63	Acute gangrenous appendicitis.	Appendectomy. No drainage.	General and novocaine.
F	60	Acute gangrenous appendicitis; pelvic peritonitis.	Appendectomy. Drainage. Extensive wound infection and sloughing of fascia.	Novocaine only.
F	14	Acute catarrhal appendicitis.	Appendectomy. No drainage.	Nitrous oxide and ether.
M	49	Acute ruptured appendicitis; generalized peritonitis.	Drainage of abdomen. Died after generalized peritonitis and pneumonia.	Novocaine and ethylene.
F	47	Acute gangrenous appendicitis.	Appendectomy. No drainage.	Novocaine and ethylene.
F	55	Acute appendicitis; peritonitis; acute diffuse appendicitis and periappendicitis.	Appendectomy. Drainage. Died after generalized peritonitis and pneumonia.	Novocaine and nitrous oxide.
F	55	Appendiceal abscess.	Drained abscess. Infected wound.	Nitrous oxide.
F	50	Appendiceal abscess.	Refused operation. Spontaneous rupture into rectum. Recovered.	No operation.

COMMENT

In some of the cases reviewed especially those admitted at night, the diabetic condi-

tion was a definite factor in causing delay in operation. Both of the fatal cases arrived at the hospital at night and operation was delayed until the next day because of the diabetic condition. Some practical points may be made. These have been gathered from the literature.

1. The aim should be to remove the infected appendix first, and to treat the diabetes later.

2. It is not necessary to wait until the urine is sugar free. Small amounts of sugar in the urine do no harm at the time of operation and depletion of carbohydrate is less likely to occur. The glycogen present in liver acts as a protective agency.

3. Blood sugar and CO_2 determinations may be done if facilities are present, but these are not imperative in an emergency, provided urine is examined for sugar, acetone and diacetic acid. Insulin is given accordingly.

4. The diacetic acid test is a reliable and satisfactory index of acidosis in surgical emergencies. It is simple to do, and can be readily repeated day or night. This test can be used as a diagnostic and therapeutic guide. If negative, operation does not have to be delayed. If positive, give 40 to 60 units of insulin and saline, and repeat the test in three to four hours. As soon as the patient is out of acidosis, operate.

5. Consultation with a medical man interested in diabetes is wise.

CONCLUSIONS

In the majority of instances of acute appendicitis in diabetes, patients come to surgery with complications of gangrene, perforation, abscess formation, or peritonitis. Delay in operation is more dangerous than in normal persons.

The diacetic acid test is a reliable and satisfactory index of the presence or absence of acidosis in acute surgical emergencies.

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THE SURGICAL TREATMENT OF ACUTE CHOLECYSTITIS*

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CAVE has aptly stated that sound judgment is required to choose the opportune moment for surgical intervention in acute cholecystitis and that any attempt to standardize the time of operation is hazardous. An early operation, as defined by Cave, implies an interval of from twenty-four hours to seven days, while a delayed procedure is one done after the acute process has subsided, a matter of weeks or even months. Likewise surgeons may be classified into three groups: "(1) those who operate immediately upon admission of the patient to the hospital (apparently regardless of the time elapsed since onset)—the "immediate" group; (2) those who operate between the first and fifth days (where apparently elapsed time since onset is given consideration)—the "early" group; and (3) those who delay intervention for weeks or even months." It is Cave's conviction that the mortality rate in the hands of the majority of surgeons is better in the so-called "early" group than in the "immediate" group.

As a starting point in the consideration of the surgical treatment of acute cholecystitis Graham's statement is singularly apropos, viz., that "the danger of operation in acute cholecystitis cannot be demonstrated in a hospital that usually practices delay, for only the bad cases will receive early operation. Conversely, a hospital that believes in early operation cannot speak with authority about the dangers of delay."

From a bacteriologic and surgical viewpoint, the findings of Edmund Andrews are of importance in this connection: (1) in many cases it is not possible to obtain positive cultures in what appears to be a very acute inflammation of the gall-bladder; (2) many cases which seem to show empyema have sterile cultures; (3) in many cases there is a stone in the cystic duct or in the ampulla, apparently slowing the blood flow from the gall-bladder.

An acute bacterial cholecystitis may not be the cause, but rather edema and obstruction of the blood supply. If these are allowed to quiet down, acute bacterial infection will develop. This is the explanation of pericholecystitis and abscess developing in the gall-bladder, producing technical difficulties in its later removal.

Burggeman drew a clear analogy when he declared that acute cholecystitis may be compared to acute salpingitis in that it rarely kills if treated conservatively. The comparison between acute cholecystitis and acute appendicitis is not in consonance with the clinical facts.

H. F. Graham, in a series of 197 consecutive cases, operated upon twenty within forty-eight hours of the onset of the acute symptoms, with one death (pancreatitis), a mortality of 5 per cent. This is, of course, a small series. In the remaining 150 cases operated upon after the forty-eight hour interval, the mortality was 6.2 per cent.

Pratt, quoted by Graham and Waters, records twenty-three cholecystectomies done within twenty-four hours of hospital entrance, without a death. The time of acute illness prior to hospital entrance and operation is not stated. In a series of forty-five cases there were no deaths when operation was done within forty-eight hours after onset of the disease.

* From the Surgical Division of the Los Angeles County Hospital and the Surgical Department of the College of Medical Evangelists, Los Angeles.

Smith reports in a series of 1,053 cases:

	Mortality
Acute, 107 cases, 10 deaths, (operated upon at once)	9.3
Subsided, (three or four days without fever before operation), 94 cases, 5 deaths, (operation from forty-eight hours to seven days after onset)	5.3
Chronic, 754 cases, 24 deaths	3.3
Duct stones, 98 cases, 32 deaths	33.0
Total number of cases 1053, 71 deaths	6.7

Five of the cases of the acute series (5 per cent) were perforated, all in the first three days after onset of the illness; eight were gangrenous; eleven empyemas were found; and eleven had abscesses located outside the gall-bladder. In several patients more than one of these complications was present. In the preceding thirty-three patients (one-third of the acute group) there occurred five deaths, one-half of the total. Thus since all fulminating cases (the thirty-three complicated cases) were included in the mortality statistics of the acute group these figures are not entirely accurate. If one deducts the thirty-three complicated cases from the 107 cases listed, there remain seventy-four strictly acute cholecystitis cases in which there were five deaths, 6.5 per cent mortality.

Smith, despite the trend toward early surgery, and certain contrary opinions, believes that ectomy in acute cholecystitis is a definitely more difficult technical procedure and states that in such cases it may be necessary to remove the gall-bladder at the ampulla rather than at the duct, as a matter of safety. Two of the cases in his series were reoperated for stones present in the remnant of the gall-bladder. Smith in a recent reversal of opinion, now believes that surgery in acute cholecystitis is unwise unless the evidence points to free perforation, in which instance immediate operation should be done.

Graham compiled a list of 153 cases of acute cholecystitis from the wards of the Toronto General Hospital for the period from July, 1, 1926 to January 1, 1934. Of these 153 cases, twenty-four refused surgery and in sixty-one surgery was not advised for various reasons. Thus of sixty-eight operated cases six were operated upon at once with one death, 16 per cent mortality. In sixty-two cases delayed surgery was done, with three deaths, 4.8 per cent mortality. Four deaths occurred in the sixty-eight cases, a mortality of 5.8 per cent. The average time from onset to hospital entrance was six days.

In a series of fifty-two private patients, Graham operated immediately in seven cases and delayed surgery in forty-five. There were four fatal cases in the series, a mortality of 7.7 per cent. In the delayed surgery group of forty-five, there were eleven perforations; all of these patients survived. These cases were seen earlier after onset than those of the series of Toronto General Hospital.

Pennoyer studied 300 consecutive cases of clinically acute cholecystitis, at the Roosevelt Hospital, New York City, the cases dating back to 1918. The routine treatment had been to delay surgery in such cases until the acute symptoms subsided, operation being done only if urgent complications indicated such intervention. During more recent years, early surgery has been advocated, the reason for the change being that the danger of complication was considered greater than the danger of operative interference. Operation is now done within five days of onset unless there is definite improvement. Cholecystostomy rather than cholecystectomy is done, on the basis of safety.

In this series, the charts of 1,474 patients with gall-bladder disease were studied. The 300 cases of acute cholecystitis presented here represent a careful selection including those in which the advocates of early operation have presented evidence of justified intervention.

There were thirty deaths in the entire series, a mortality of 10 per cent. In these fatal cases, the average time of acute illness prior to hospital entrance was four days; they were observed for two and one-third days prior to operation. Thus a total of

six and one-third days represents the average time from onset to operation. Of these thirty deaths, twenty-one actually died as a result of operative procedure, a 7 per cent mortality. Fifteen deaths occurred in a series of fifty-nine cases (20 per cent of the entire series of 300), in which emergency operation was done within twelve hours of hospital entrance. In this series of fifty-nine, there were thirty-two cases, 54 per cent, incorrectly diagnosed. Thus, the fifteen deaths, operated at once as emergencies, represent a mortality of 25 per cent.

The remaining 241 patients had been acutely ill for four days prior to hospital entrance. Of these, thirty-three, after an average delay of five days, were found not subsiding or actually becoming worse. They were therefore operated upon in the presence of acute symptoms, seven deaths ensuing, a mortality of 20 per cent.

In the remaining 208 cases the symptoms subsided after an average of eight days. Thus the total time from onset to operation in these 208 cases was at least twelve days. There were five deaths in this group, a mortality rate of 2.5 per cent.

In the entire group of 241 cases there were twelve deaths, a mortality of 5 per cent.

Of the 300 cases, 20 per cent showed some gangrene of the gall-bladder wall and thirty cases showed definite rupture with resultant local peritonitis.

Cutler, in the discussion of Pennoyer's paper, insisted that only cases seen within a few hours of the onset of the attack, not two to five days after onset, should be operated upon at once. Whipple is of the same opinion. Leidberg believes that moderately severe cases of acute cholecystitis should be operated upon in two or three days, unless specific contraindications are present.

Branch and Zollinger (235 cases) performed immediate surgery in thirty-four cases, the mortality being 20.3 per cent. In 195 cases they waited an average of 4.7 days before surgery; operative mortality was reduced to 8.7 per cent.

Heuer, in a summary of 1,066 cases of acute cholecystitis subject to surgery, found a general mortality of 8 per cent and an individual mortality varying between 4.7 per cent to 22.5 per cent. The report of Judd and Phillips, concerning 508 cases of acute cholecystitis, showed a mortality percentage of 4.7. There were sixty-eight cases of perforation or gangrene, 13 per cent. Cave comments that only fourteen of these 508 patients had emergency operations. Also to be noted is the fact that of the sixty-eight cases having gangrene or perforation, sixty-one had the lesion walled off into a localized abscess. Cave has the definite conviction that the acutely inflamed gall-bladder rarely perforates into the free abdominal cavity, causing a diffuse peritonitis, but that the percentage of localized abscess following perforation is quite large.

Eliason and McLaughlin report nine perforations in a series of 500 consecutive hospital admissions for biliary disease, a percentage of 1.8. All had localized abscess, six being admitted as emergency cases. Inclusive of their own 500 cases, they record a summary of 7,316 cases; ninety-six instances of perforation occurred, 0.77 per cent. Perforation in the cases of the different surgeons of this series ranged from 0.9 to 2.5 per cent, mortality from 11 per cent (Eliason and McLaughlin) to 44 per cent.

Touroff found that 20 per cent of seventy-five cases of acute cholecystitis, in the presence of minimal or absent clinical manifestations, had progressive gall-bladder lesions. The remaining 80 per cent (sixty cases) had lesions that were subsiding or capable of subsidence. Touroff believes that if subsidence does not occur promptly and proceed regularly, early operation is indicated. In cases with subsided clinical manifestations, early operation, rather than later in the "interval" is indicated.

Shoemaker reviewed 274 operations upon the biliary tract, done by staff surgeons of the Los Angeles County Hospital from 1929 to 1931. These patients were selected from 1,186 admitted to the hospital with a diagnosis of acute or chronic cholecystitis. Of

the number of patients admitted, approximately 25 per cent were operated upon. Of these, thirty-seven died, a mortality of 13.5 per cent. Discarding the six malignancies in the series, the mortality becomes 11.3 per cent. There were two cases of acute ulcerative cholecystitis, one abscess of the liver, two gangrenous gall-bladders and four empyemas in this series.

In this survery covering all biliary cases admitted to the Los Angeles County Hospital for the five-year period, 1933–1938, we have been particularly interested in attempting to determine how much influence, if any, the elapsed time from the onset of an acute cholecystitis to operation is related to complications and mortality, the criterion to be the pathologic diagnosis rather than the surgeon's operative report, in all cases where cholecystectomy was done. In those instances where cholecystostomy was done, the surgeon's operative report was used, unless a portion of the gall-bladder had been exised and used for pathologic examination.

Questionable cases in the original list have been discarded whenever there was insufficient data. A total of 131 cases were thus excluded. These would probably have belonged in Group 1 or 2. Likewise all cases of primary pancreatic disease and all malignancies of the biliary tract (sixty-six) have been excluded. It has been impossible to tabulate the cystic duct stone cases separately because of lack of data. They are therefore included in the general tables. We are of the opinion that the importance of the cystic duct stone cases, as related to vicious complications and resultant increased mortality, has not been sufficiently stressed.

The universal policy of this hospital has been to defer surgery until the acute attack has subsided, subject to the indications for emergency operations at the discretion and judgment of the attending surgeons. It is uncommon for the acute case to enter the hospital within forty-eight hours of the onset. The patient has been ill usually from two to seven days before hospital entrance. The suggestion of some observers that operation be done in the first forty-eight hours is limited in its applicability in this hospital.

There were 955 patients admitted with a diagnosis of biliary disease in the period 1933–1938. This includes all questionable biliary and malignant cases, which were excluded from our final tables. In the entire group, there were 704 surgical interventions.

Acute cholecystitis was diagnosed in 251 patients. Six cases are excluded because of insufficient data, leaving 245. None of this group was operated upon; either the patient refused surgical intervention or the surgeon objected, usually on the basis of other additional and advanced pathology, especially cardiorenal disease.

There were fifteen deaths in this nonoperative group, a mortality of 6 per cent. Nine autopsies were done, and in all but two instances, the cause of death was other than acute cholecystitis or its complications. However, all autopsies showed the presence of cholecystitis.

Since 230 of these 245 unoperated patients apparently recovered and were dismissed from the hospital in from five to fourteen days, there is presumptive evidence that the incidence of perforation, gangrene and abscess may not be so high as some observers report.

The operative cases have been tabulated in three groups: Group 1 represents all cases operated upon within forty-eight hours of the onset of the illness. Group 2 includes those cases operated upon from the third to the sixth day inclusive after onset, and Group 3 those cases operated upon after the sixth day. All questionable cases (197) have been excluded.

Group 1. Operation done within forty-eight hours of onset. There were sixteen cases in this group, eight cholecystectomies and eight cholecystostomies, with three deaths, a mortality of 19 per cent.

There were five cholecystectomies in females, no deaths; three in males with one death: the mortality thus being 12.5 per

cent. Pathologic diagnoses were: two subacute, one marked subacute, one empyema, one gangrene and three chronic cholecystitis.

Table I
GROUP I
(Operated upon within 48 hours of onset)

Sex	Type of Operation	No.	Deaths	Acute	Subacute	Chronic	Gangrene	Empyema	Hydrops
F	ect.	5	0	0	1	2	1	1	
M	ect.	3	1	0	2	1	0	0	
F	ost.	4	1	..	..	..	..	..	1
M	ost.	4	1	..	..	..	..	1	

Total, 16 cases, 3 deaths, mortality, 19 per cent
8 ectomies, 1 death, mortality, 12.5 per cent
8 ostomies, 2 deaths, mortality, 25 per cent

The eight cholecystostomies were evenly divided as to sex. There were two deaths, a male and a female, giving a mortality of 25 per cent. One hydrops and one empyema were included.

Group 2. Operated upon from forty-eight hours to the seventh day after onset. There were sixty-four cases in this group, with

Table II
GROUP 2
(Operated upon from 48 hours to the 7th day after onset)

Sex	Type of Operation	No.	Deaths	Acute	Subacute	Chronic	Ulcerative	Gangrene	Empyema	Hydrops
F	ect.	36	2	4	7	19	4	2	0	0
M	ect.	12	2	3	2	5	1	0	0	1
F	ost.	11	0	..	1	..	..	1	1	
M	ost.	5	1	..	..	..	..	1	1	

(Ulcerative, 1 acute, 1 recent acute, 1 subacute, 2 chronic)
Total, 64 cases, 5 deaths, mortality, 7.8 per cent
48 ectomies, 4 deaths, mortality, 8.3 per cent
16 ostomies, 1 death, mortality, 6.25 per cent

five deaths, a mortality of 8 per cent. Forty-eight cholecystectomies were done, with four deaths, a mortality of 8.3 per cent. There were thirty-six in females (2 deaths), twelve in males (two deaths). The pathologic diagnosis were: seven acute, nine subacute, five ulcerative, two gangrenous, one hydrops and twenty-four chronic. The five ulcerative cases were further classified as: one recent acute, one subacute, one acute and two chronic.

Sixteen cholecystostomies were done with one death, a mortality of 6.2 per cent. In eleven females there were no deaths. The pathology was given as one subacute, one gangrene and one empyema. In the five males there was one death.

Group 3. Operated upon after the sixth day since onset. The greater portion of this

Table III
GROUP 3
(Operated upon after the 6th day after onset)

Sex	Type of Operation	No.	Deaths	A	S-A	Chr.	Pur.	Ulcer.	Perf.	Gang.	Emp.	Hyd.	Chol.	No report
F	ect.	311	13	8	58	208	4	21	0	0	0	1	1	10
M	ect.	79	7	3	18	45	0	7	0	1	1	0	0	4
F	ost.	27	1	..	..	..	..	..	..	..	3			
M	ost.	10	2	..	..	1	..	..	2	1	3			

(Purulent, 1 recent acute, 2 subacute, 1 chronic)
(Ulcerative, 4 acute, 1 recent acute, 8 subacute, 15 chronic)
Total, 427 cases, 23 deaths, mortality, 5.4 per cent
390 ectomies, 20 deaths, mortality, 5.1 per cent
37 ostomies, 3 deaths, mortality, 8.1 per cent

group was operated on from the fourteenth to the twenty-first day after onset, the majority on the fourteenth day. Cases of perforation, gangrene, empyema, acute ulcerative and purulent were operated upon between the seventh and fourteenth days after onset. (Table III.)

Table IV
LOS ANGELES GENERAL HOSPITAL
1933–1938

	Per Cent Mortality
704 Surgical interventions	13.35
638 Biliary surgery solely	8.77
507 Corrected list, biliary surgery	
Group 1	19.0
Group 2	7.8
Group 3	5.4

This group comprises 427 cases, with twenty-three deaths, a mortality of 5.4 per

cent. There were 390 cholecystectomies with twenty deaths, a mortality of 5.1 per cent. In this group 311 were females, including thirteen deaths. The pathologic diagnoses in these 311 cases included: eight acute, fifty-eight subacute, 208 chronic, one cholesterosis, one gangrene, one hydrops, four purulent, and twenty-one ulcerative. The ulcerative cases were further classified as: acute ulcerative three, subacute seven and chronic eleven. The purulent were also further classified as acute one, subacute two and chronic one. No report was given in ten instances.

There were seventy-nine cholecystectomies in males, with seven deaths. Three were acute, eighteen subacute, forty-five chronic, one empyema, one gangrene and seven ulcerative; the latter classified as: acute one, subacute one, recent acute one, and four chronic. No report was given in four cases.

Thirty-seven cholecystostomies were done, twenty-seven in females and ten in males, with three deaths, a mortality of 8.1 per cent. There was one death in a female. Three empyemas were included. In the ten males two deaths occurred. One gangrene, two perforations, three empyemas and three chronic cholecystitis were present.

SUMMARY

We are in accord with Smith in the belief that operation within forty-eight hours of onset carries too high a mortality to warrant much consideration, especially in the wards of a public charitable hospital whose patients are not infrequently poor surgical risks. Furthermore, these patients often do not come to the hospital within forty-eight hours of the acute onset. Our opportunities have been limited as regards statistics for this group. Our entire Group 1 series has little positive value since it is too small in numbers.

We are inclined to the view of Cutler and Whipple that any early operation, preferably cholecystectomy, should be done within six to twelve hours of onset. So far as we have been able to determine, apparently the peak of the disease is reached in from one to four days.

In Group 2, after forty-eight hours, seven acute, ten subacute, five ulcerative, four gangrenous, two empyemas and one hydrops were present; in other words, considerable acute pathology remained. The chronic cases, almost a third, showed subsidence of the pathology. Nevertheless the mortality for this group had dropped by 60 per cent, from the 19 per cent of Group 1 to 7.8 in Group 2.

In Group 3, in which operation occurred after six days (most of them about the fourteenth day), we still find evidence of acute gall-bladder pathology. There was evidence of an active pathology in 130 cases out of 427 supposedly subsided cases. In all of these we waited, and in the majority of instances they showed minimal clinical manifestations when operated upon. Yet acute lesions were present in 31 per cent of cases. The mortality, however, dropped from 19 per cent in Group 1 and 7.8 per cent in Group 2 to 5.4 per cent in Group 3.

There were nine deaths in Group 3 due to accidental injuries of ducts and blood vessels. These deaths represented almost 40 per cent of the total mortality of this group; if these nine deaths were eliminated the mortality would be 3.5 per cent.

COMMENT

The very frequent occurrence of acute pathology in Groups 2 and 3 does much to influence our belief that no absolute time can be set as to the opportune moment to operate.

We are, however, firmly convinced of the verity of Touroff's observations, that advanced pathology may be present with minimal clinical manifestations and that each case should be conducted on a knowledge of Touroff's pertinent facts and be sharply individualized.

We are of the opinion that, except for those cases of perforation and gangrene which may apparently occur at any and all times, the mortality percentage will probably be lowest when operation is done about fourteen days after onset.

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Non-lithogenous cholecystitis occurs in 10 per cent of cases. As has been previously indicated, the infection is of a haematogenous origin, and is caused by a particular strain of streptococcus which exhibits a predilection for the wall of the gall-bladder.
From—"Surgery of the Alimentary Tract" by Devine (Williams & Wilkins Company).

MECKEL'S DIVERTICULUM*

WILLIAM W. NOEL, M.D., F.A.C.S.

DETROIT, MICHIGAN

TWENTY-FIVE Meckel's diverticula have been observed and described at operations in the Henry Ford Hospital during the past thirteen years. They present such a variety of findings that their study should be of interest.

Meckel's diverticulum is described as an occasional sacculation or cecal appendage of the ileum, derived from an unobliterated vitelline duct. It is therefore a congenital anomaly due to a defect in the closure of the omphalomesenteric duct. It is a rather common anomaly; various reports of autopsy findings place its incidence at from 1.5 to 2.5 per cent. The twenty-five cases here recorded were found among approximately 12,000 intra-abdominal operations. It was thus observed in only 0.21 per cent of these patients and must have occurred in many more. Until recently it has not been searched for, unless the symptoms and findings were not explained by other pathologic processes.

Age seems to have played very little part in the discovery of these diverticula. (Table I.) Seventeen of the twenty-five, or 69 per cent, occurred between the ages of 20 to 50 years. Extremes of life were present in a baby of fourteen weeks and a man 77 years of age. Sex seems to play a definite rôle. There were fifteen males and ten females in this series and all were of the white race.

TABLE I

Ages	Number
1–10	4
11–20	1
21–30	5
31–40	5
41–50	7
51–60	2
77	1

Morbid Anatomy. The diverticula vary markedly in size, shape, position and structure. The majority in this series were approximately the size of a thumb. (Table II.) The extremes varied in size from a sac less than 2.5 cm. long to one more than 33 cm. in length. The diameter was more constant, averaging 1 to 1½ inches. There were many different shapes. One was called conical, two bulbous, two tapering and one had a forked end. One had a sphincter-like mechanism at its neck.

TABLE II

Number	Length, Cm.	Diameter, Cm.
2	2.5	
2	5.0	
3	5.0	2.5
1	5.0	4.0
1	6.0	
1	6.0	1.0
1	6.0	4.0
2	7.5	2.5
1	7.0	4
1	11.0	4
1	12.5	2
1	Small	
2	Size of distal phalanx	
4	Size of thumb	
1	Size of egg	
1	33 cm. long, size of intestine	

Nine of the diverticula were described as anti-mesenteric in position, three were definitely associated with the mesentery and the exact position of the others was not mentioned. The relation to the ileocecal valve is shown in Table III. In this series they were situated from 2 inches to 3 feet from the cecum.

TABLE III

POSITION IN RELATION TO ILEOCECAL VALVE

Number	Distance, Inches
4	36
4	24
1	20
8	18
2	10
1	2
5	Not mentioned

* From the Department of Surgery, Henry Ford Hospital.

The majority of the diverticula lay free except for the attachment to the ileum. One was attached to the umbilicus, one had diverticulum occurred in the mesentery just 2 inches from the cecum. It was called acquired by the operating surgeon because

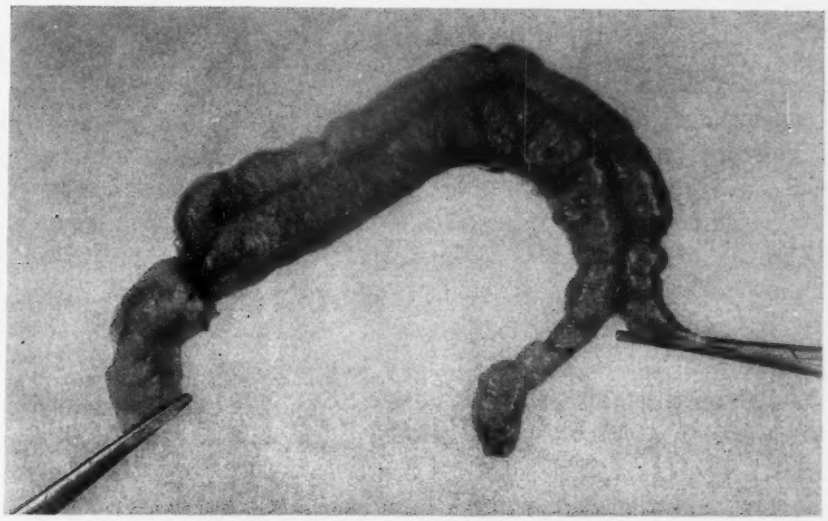

Fig. 1. Giant diverticulum. Clamp on each end of ileum. Perforation at junction of diverticulum and ileum.

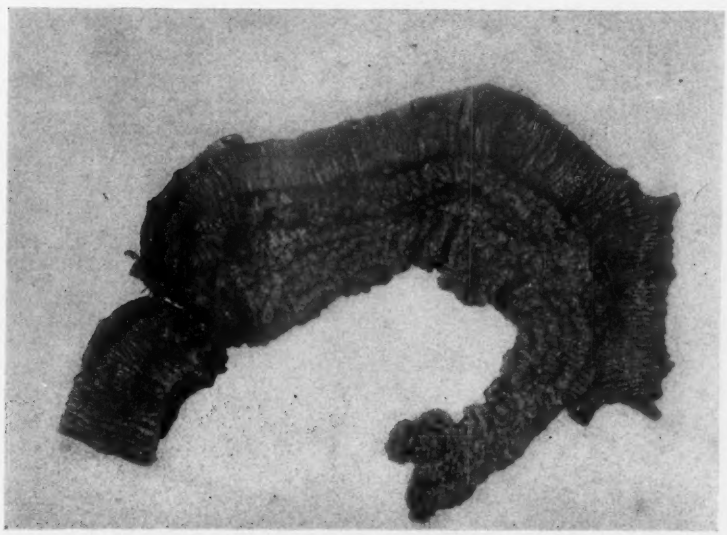

Fig. 2. Ileum and diverticulum open. Note gastric mucosa.

the tip buried in the mesentery, four were bound to the mesentery by adhesions. The giant diverticulum, in close association with the ileum and its mesentery, presented the appearance of a double-barrelled portion of the intestine. The diverticulum and intestine had a common mesentery except at the distal end of the diverticulum where it possessed a separate portion. (Figs. 1 and 2.)

Three diverticula in all were closely associated with the mesentery. The smallest of its position. It contained, however, all the usual layers of the bowel wall and is therefore classified here as congenital. The third one occurring in close relation to the mesentery proved to have a short mesentery of its own when the peritoneum over it was incised.

An acute inflammatory process was described in two cases and in three others ulceration was present. Unfortunately, amputation had to be done at the site of the ulceration in one case and enterostomy was

made at this site in another case, so the ulcers were not demonstrated pathologically. The ulcer in the last case had perforated at the time of operation. The third diverticulum had the gastric mucosa limited to the tip with an ulcer located in the center of this. (Fig. 3.)

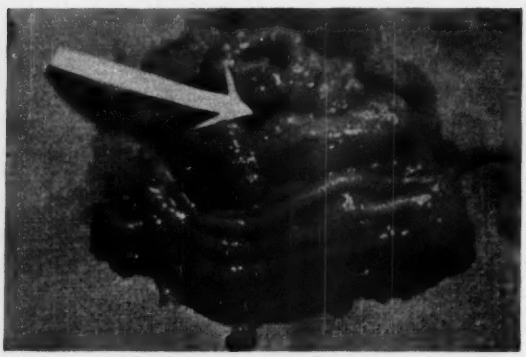

Fig. 3. Diverticulum opened. Note gastric mucosa and ulcer (arrow pointing to ulcer.)

Intestinal obstruction was present in six cases. Adhesions binding the diverticulum to the mesentery was the cause in four cases. In one instance the obstruction occurred about a large mesentery to the diverticulum and in the other a portion of ileum passed through a loop between the mesentery and the diverticulum, the tip of which was attached to the mesentery.

Hemorrhage from the bowel was present in three instances and from the umbilicus in a third case.

Foreign bodies in a diverticulum are rather rare. In one of these cases a bullet was present and in another many small bodies which suggested gallstones. (Fig. 4.) An analysis of one of these bodies revealed that they were composed of dried intestinal contents.

Associated anomalies occurred in three cases. The gall-bladder and cystic duct were proved to be absent at post-mortem in the man of 77. A very large tumor of the right pleural cavity, presumably a dermoid, was present in the baby with the giant diverticulum. A small cord, the remnant of the allantois was present in the patient whose diverticulum was attached to the umbilicus.

Histopathology. Sixteen specimens were submitted to microscopic examination. Thirteen of these were described as normal Meckel's diverticula, without inflammation, two were described as acutely inflamed and one presented edema. Gastric mucosa was present in three, duodenal mucosa in two and upper jejunal mucosa in another. (Figs. 4, 5 and 6.) The aberrant mucosa was found in the recent cases and was un-

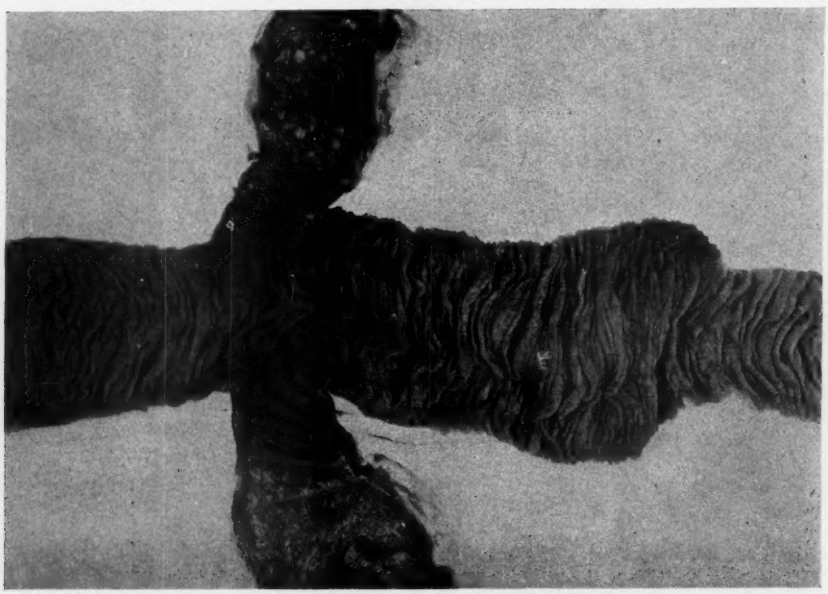

Fig. 4. Foreign bodies in a Meckel's diverticulum. The ileal mucosa in this case is acutely inflamed.

doubtedly present in some of the others. The giant diverticulum was lined throughout by gastric mucosa.

Symptoms. Thirteen of the patients presented no symptoms referable to the diverticulum. These patients were operated on for other conditions and the diverticulum was an incidental finding.

The other twelve patients presented symptoms directly attributable to the diverticulum or to complications resulting from its presence. The symptoms in these cases were variable depending on the pathologic involvement of the diverticulum. Six were associated with the symptoms of intestinal obstruction. There were no previous symptoms suggesting the presence of the Meckel's diverticulum. Two patients had symptoms suggestive of appendicitis and four had bleeding as a cardinal symptom. This occurred from the umbilicus in one case and from the bowel in three.

have been conjectured in at least two others, one with bleeding from the umbilicus and another in which there was the

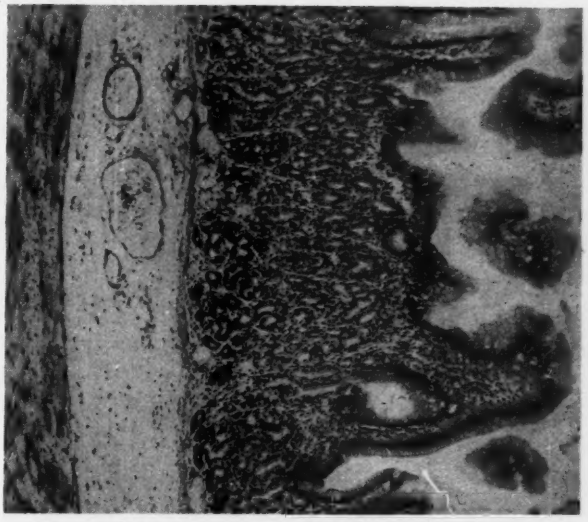

Fig. 5. Gastric mucosa in Meckel's diverticulum.

unusual history and finding of a foreign body, presumably a bullet, occurring frequently in a right scrotal hernia. This last patient had been shot through the abdomen some years before. The fact that the bullet was found in the hernial sac was proof that it must have been in an intestinal diverticulum or possibly in the omentum. In six patients with intestinal obstruction the diagnosis was made preoperatively in four.

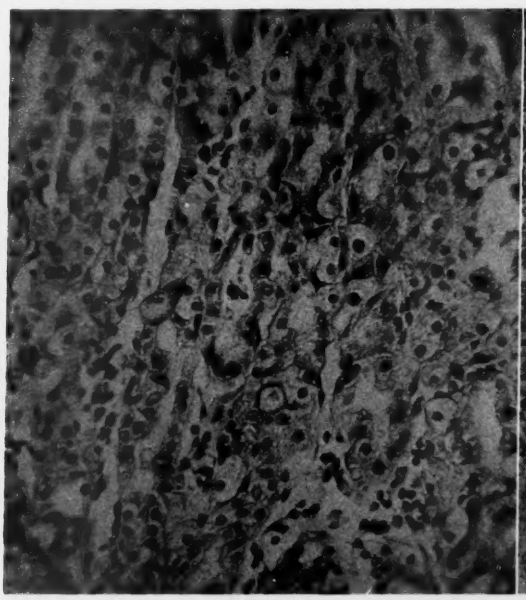

Fig. 6. High power magnification of a portion of gastric mucosa shown in Figure 4.

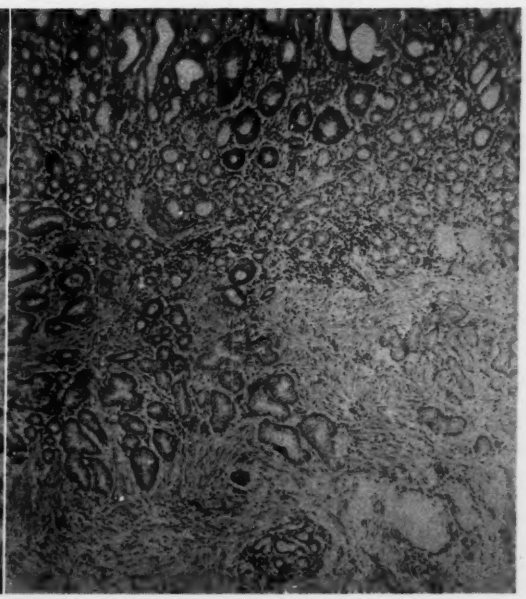

Fig. 7. Duodenal mucosa in a Meckel's diverticulum.

Diagnosis. The preoperative impression was correct in only three of the cases as described. (Table IV.) The diagnosis could

Table IV

Diagnosis	No.
Intestinal obstruction	6
Appendicitis	2
Meckel's diverticulitis (with bleeding)	3
Patent urachus (with bleeding)	1

The other two were diagnosed as appendicitis and obstruction to the cystic duct. The last patient was jaundiced, apparently due to some obstruction about the cystic duct which had been anastamosed to the duodenum some time before. The intestinal obstruction also played some part in the obstruction to the cystic duct. An operative diagnosis of Meckel's diverticulitis was made in one case where symptoms suggested appendicitis, and in another where intestinal obstruction had been considered probable. Despite the fact that the findings were those of an acute abdomen and the white blood counts were 15,000 and 12,000, respectively, two diverticula, called acute at operation, showed only mild edema in one and entirely normal structure in the other.

A rather typical history and findings made possible the correct diagnosis in one patient aged 7 years. Five weeks before admission there were abdominal cramps followed by a grossly bloody stool and a feeling of faintness. Anorexia persisted until the day before admission, when there was nausea associated with abdominal pain. The findings of right lower quadrant tenderness, spasm and rebound tenderness, associated with a white blood count of 9,200 and 81 per cent polymorphonuclear cells suggested the diagnosis of Meckel's diverticulitis. In a patient with frequent hemorrhages from the bowel and abdominal patterns suggesting some degree of obstruction, and in another patient who reported two episodes of profuse intestinal bleeding associated with nausea, there was even less difficulty in diagnosis.

Treatment. The treatment is always surgical. The pathologic process resulting as a complication of the diverticulum must be given first attention, but the diverticulum should usually be removed.

The diverticula here reported were seen by seven different surgeons. Treatment was of five different types. (Table v.) The diverticulum was inverted in a case of intestinal obstruction, presenting a greatly dilated bowel and a dilated, thin-walled, wide-mouthed diverticulum, obstructed about a large mesentery to the diverticulum. The mesentery with its large vessels was divided and a simple inversion carried out, the bowel being brought together transversely with interrupted sutures of silk. It was felt that the diverticulum would certainly slough. A simple enterostomy, with a mushroom catheter, was made about 18 inches above this. The catheter was held in the bowel with a purse-string suture of silk, the free end being drawn through a stab wound in the abdominal wall, thus approximating the bowel to the peritoneum. The enterostomy closed promptly after removal of the tube on the fourteenth postoperative day.

Table v

METHOD OF TREATMENT OF THE DIVERTICULUM

Excised in the manner of an appendix.	13
Inverted with enterostomy.....	1
Resected along with portion of ileum.	5
Exteriorized with portion of bowel...	1
Left undisturbed.....	5

The diverticula were small-necked sacs in thirteen cases; these were treated as we would treat an appendix. A purse-string suture was placed about the base of the diverticulum and sterile gauze applied around it. The peritoneum was incised about 0.5 cm. above the purse-string, this area was crushed with a small clamp, and the neck ligated with catgut. A Halsted clamp was placed distal to the ligature and the diverticulum divided. The stump was carbolized and inverted through the purse-string suture, which was drawn taught. An improvement in technique at this point is to place several interrupted Lembert sutures to strengthen the inversion, before the purse-string suture is tied, in this way avoiding the protrusion of the stump that sometimes occurs when the purse-string is inadvertently broken. This treatment of the diverticulum is the method of choice in most instances.

The bowel was resected in five cases. This was deemed to be necessary in three cases of intestinal obstruction, in one case of terminal ileitis and in one case in which the bowel and diverticulum possessed a mesentery in common. The resection usually

consisted in removing a short piece of ileum on each side of the sac, with a lateral anastomosis completing the operation.

The diverticulum was exteriorized along with the gangrenous bowel in the case of the strangulated hernia. It had nothing to do with the strangulation.

Mortality. Two patients in this series died. The 77 year old patient died three days after operation. A woman of 56 died from intestinal obstruction associated with a strangulated hernia. Only twenty of the twenty-five cases had any form of treatment directed to the diverticulum so this would give a mortality rate of 10.0 per cent. The prognosis is good for complete recovery after the diverticulum is removed.

CASE REPORTS

Asymptomatic Group. 1. G. G. Male, white, 46 years old. Bleeding duodenal ulcer. Posterior gastroenterostomy, excision of diverticulum. Pathology—Meckel's diverticulum. Recovery.

2. M. H. Female, white, 26 years old. Chronic appendicitis. Appendectomy, excision of Meckel's diverticulum. Pathology—acute Meckel's diverticulitis. Recovery.

3. E. B. Female, white, 28 years old. Salpingitis. Salpingectomy, appendectomy, excision of Meckel's diverticulum. Pathology—Meckel's diverticulum. Recovery.

4. S. V. Male, white, 43 years old. Terminal ileitis with abscess. Resection of terminal ileum and diverticulum, appendectomy. Pathology—Meckel's diverticulum, duodenal mucosa. Recovery.

RESULTS AND FOLLOW-UP

	Treatment	Last seen	Result
Asymptomatic group associated with:			
Bleeding duodenal ulcer	Resected	8 years	Well. (Later resection ulcer.)
Chronic appendicitis	Resected	3 years	Well
Salpingitis	Resected	10 years	Primary anemia
Hernia	Resected	1 month	Well
Strangulated hernia	Exteriorized	24 hours	Expired
Terminal ileitis	Resected ileum	1 year	Well
Recurrent appendicitis	Resected	2 months	Well
Salpingitis	Resected	1 month	Well
Symptomatic group associated with:			
Intestinal obstruction	Resection ileum	13 months	Well
Intestinal obstruction	Resected	3 days	Expired
Intestinal obstruction	Resected	5 years	Well
Intestinal obstruction	Inverted	24 months	Well
Intestinal obstruction	Resection ileum	6 months	Well
Intestinal obstruction	Resection ileum	5 months	Well
Associated with hemorrhage from:			
Umbilicus	Resected	1 month	Well
Bowel	Resected	7 months	Well
Bowel	Resection ileum	18 months	Well
Bowel	Resected	1 month	Well
Acute diverticulitis:			
First	Resected	1 month	Well
Second	Resected	2 months	Well

The follow-up has been inadequate, but it is to be noted that none of the patients has returned for treatment for a cause in any way associated with the diverticulum. This is especially noteworthy since in five cases the diverticulum was not disturbed.

5. M. C. Female, white, 30 years old. Recurrent appendicitis. Appendectomy, excision of Meckel's diverticulum. Pathology—Meckel's diverticulum. Recovery.

6. J. W. Male, white, 35 years old. Gunshot wound in abdomen, 1929. Right inguinal hernia with bullet palpable in sac at times. Hernioplasty, excision of Meckel's divertic-

ulum, appendectomy. Pathology—Meckel's diverticulum. Recovery.

7. E. S. Female, white, 56 years old. Strangulated inguinal hernia, right, with Meckel's diverticulum in the sac. Exteriorization of the bowel and diverticulum. Expired in twenty-four hours.

8. I. B. Female, white, 38 years old. Salpingitis. Release of adhesions, excision of corpus luteum cyst, excision of Meckel's diverticulum, appendectomy. Pathology—Meckel's diverticulum. Recovery.

Intestinal Obstruction Group. 9. B. E. Female, white, 38 years old. Generalized pain, nausea and vomiting for thirty-six hours. Previous appendectomy and cholecystectomy. Acutely ill, distended R.L.Q. tenderness and spasm. *X*-ray-dilated coils. W.B.C. 10,800; 83 per cent polys. Intestinal obstruction about a swollen, adherent Meckel's diverticulum. Resection of 10 inches of ileum with diverticulum. Pathology—Meckel's diverticulitis with multiple concretions. Recovery.

10. G. P. Male, white, 77 years old. Generalized pain, nausea and vomiting for two days. Ill, distended, R.L.Q. tenderness and spasm. W.B.C. 9,500, 72 per cent polys. Diagnosis—intestinal obstruction about a Meckel's diverticulum involved in adhesions. Release of obstruction, excision of diverticulum. Pathology—Meckel's diverticulum. Expired in three days. Autopsy, absent gall-bladder and cystic duct.

11. A. K. Female, white, 46 years old. Paraumbilical pain, nausea, and vomiting of four days' duration. Moderately ill, distended, generalized tenderness. W.B.C. 7,700. Diagnosis—intestinal obstruction about mesentery of Meckel's diverticulum. Release of obstruction, division of mesentery, inversion of diverticulum and enterostomy. No specimen. Recovery.

12. H. N. Male, white, 21 years old. Generalized pain, nausea and vomiting of two days' duration. Ill, generalized and R.L.Q. tenderness and spasm. W.B.C. 8,900, 78 per cent polys. Impression—probably appendicitis. Diagnosis—intestinal obstruction, loop of bowel between diverticulum and mesentery of ileum to which it was bound. Release of obstruction, excision of Meckel's diverticulum. Pathology—Meckel's diverticulum. Recovery.

13. R. S. Male, white, 47 years old. R.U.Q. pain, nausea and vomiting, jaundice, recurrent. Ill, R.U.Q. tenderness. Impression—obstruction to a transplanted cystic duct. Diagnosis—partial intestinal obstruction about a Meckel's diverticulum bound down by adhesions. Resection of 4 inches of ileum with the diverticulum. Pathology—Meckel's diverticulum with sphincter-like neck, duodenal mucosa. Recovery.

14. J. T. Male, white, 37 years old. Epigastric pain, nausea and vomiting of three days' duration. Not acutely ill, epigastric tenderness. W.B.C. 9,900, 72 per cent polys. *X*-ray suggested terminal ileitis. Impression of terminal ileitis with obstruction. Diagnosis—intestinal obstruction about a Meckel's diverticulum involved in adhesions. Resection of portion of ileum with Meckel's diverticulum. Pathology—Meckel's diverticulum, gastric mucosa. Recovery.

Acute Diverticulitis Group. 15. M. B. Male, white, 15 years old. R.L.Q. pain, nausea and vomiting of six hours' duration. Ill, R.L.Q. tenderness and spasm. W.B.C. 14,950, 93 per cent polys. Impression—acute appendicitis. Diagnosis—Meckel's diverticulitis. Excision. Pathology—edema of Meckel's diverticulum. Recovery.

16. C. G. Male, white, 22 years old, Generalized pain, nausea and vomiting of five days' duration. Ill, right sided tenderness and spasm. W.B.C. 12,000, 82 per cent polys. Impression—intestinal obstruction. Diagnosis—Meckel's diverticulitis. Excision of Meckel's diverticulum and appendectomy. Pathology—Meckel's diverticulum. Recovery.

Group with Bleeding. 17. E. S. Male, white, eight months old. Fistula at umbilicus since birth. Profuse bleeding from umbilicus at six and seven months. Impression—patent urachus. Diagnosis—persistent omphalomesenteric duct, remains of urachus. Excision of Meckel's diverticulum and fibrous cord. Pathology—Meckel's diverticulum. Recovery.

18. J. K. Female, white, 7 years old. R.L.Q. pain and nausea of two days' duration. Abdominal cramps associated with rectal bleeding five weeks before. Ill, R.L.Q. tenderness and spasm. W.B.C. 9,200, 81 per cent polys. Impression—Meckel's diverticulitis. Diagnosis—Meckel's diverticulitis with ulceration. Excision. Pathology—Meckel's diverticulum with upper jejunal mucosa. Recovery.

19. L. McR. Male, white, fourteen weeks old. Hemorrhage from the bowel at eight weeks and at fourteen weeks. Abdominal patterns. W.B.C. 22,000, 52 per cent polys, R.B.C.

1,800,000. Impression—probable Meckel's diverticulitis. Diagnosis—giant Meckel's diverticulitis with ulceration and perforation. Enterostomy and later resection portion of ileum and diverticulum. Pathology—Meckel's diverticulum with gastric mucosa. Associated finding probable dermoid cyst of right pleural cavity.

20. G. T. Male, white, 9½ years old. Hemorrhage from the bowel at 8½ years. Some nausea followed by bloody stool prior to admission. Abdomen essentially normal. R.B.C. 2,650,000, Hgb. 7 Gm. Impression—ulcer of Meckel's diverticulum. Diagnosis—Meckel's diverticulum—gastric mucosa with ulceration. Excision of Meckel's—appendectomy. Pathology—Meckel's diverticulum with gastric mucosa. Recovery.

21–25.* The diverticula were left undisturbed in five cases. Cholecystectomy was performed in two of these cases, appendectomy in one, a resection of the rectum in the fourth, and release of adhesions in the fifth.

DISCUSSION

Twenty-five patients with Meckel's diverticula have been presented. There was one death in six cases with intestinal obstruction, excluding the case with the strangulated hernia in which the diverticulum played no part. This gives a mortality rate of 16.6 per cent for this group. Halsted[1] reports sixty-nine cases of intestinal obstruction associated with Meckel's diverticulum; fifty-seven of these came to operation with a resultant death rate of 59.1 per cent. He gives some interesting figures on the conditions found. Forty-eight diverticula were attached to some intraperitoneal structure; twenty-three to the mesentery, fifteen to the umbilicus, three to the small intestine, one to the mesocolon, one to the mesorectum, one to the omentum and one to the appendix. Fifteen were quite free and six were not described.

The anatomic types most frequently seen are described by Thompson[2] and those of this series are compared with his figures.

1. Located on antimesenteric side, with closed distal end—82.5 per cent. This series 80.0 per cent (probably).

2. Partially obliterated with fibrous cord to the umbilicus—10 per cent. This series none.

3. Umbilical fistula—6 per cent. This series 4.0 per cent.

4. Giant diverticula—5 per cent. This series 4.0 per cent.

5. Umbilical polyp—0.5 per cent. This series none.

6. Simple intramesenteric—0.5 per cent. This series 4.0 per cent.

Other malformations have frequently been found accompanying a Meckel's diverticulum. Christie[3] in sixty-three cases found twenty-one associated anomalies. These appeared to be most common in the brain and cord, heart, urinary tract, spleen and stomach, in the order named. Moll[4] reports a retention cyst in the left pleural cavity in a patient presenting a giant Meckel's diverticulum 33.5 inches long. Three anomalies in this series consisted of the tumor of the right pleural cavity, probably a dermoid, associated with the giant diverticulum, the absence of the gall-bladder and cystic duct in the oldest patient and the small remnant of the urachus.

Hemorrhage from a Meckel's diverticulum is usually or possibly always associated with ulcer, even though the ulcer is not always demonstrable. Achsner and Karelitz,[5] in thirty-three cases collected from the literature in 1930, found that all except five reported the passage of blood. The ulcer usually occurs at the junction of the diverticulum with the ileum. Brown and Pemberton[6] in eight cases with ulcer, noted a history of bleeding from the bowel in 100 per cent, and all these diverticula were

* Dr. A. G. Lambert has recently had a further case in the hospital. A. T. Male, white, 38 years old. Generalized abdominal pain of two days' duration. Frequent vomiting. A similar attack one month before associated with diarrhea. Amebic dysentery in 1935. Ill, generalized abdominal tenderness and muscle spasm. Moderate distention. W.B.C. 4,400, 44 per cent polys. Impression—intestinal obstruction of small bowel. Diagnosis intestinal obstruction about mesentery of a Meckel's diverticulum, to which the omentum was densely adherent. Release of obstruction and resection of diverticulum. Pathology—Meckel's diverticulum, ileal mucosa with ulceration. Course—satisfactory.

lined with gastric mucosa. Gastric mucosa was present in only two of the four cases in this series presenting bleeding as a cardinal symptom. Upper jejunal mucosa was found in the third and normal ileal mucosa in the fourth case. One patient with typical gastric mucosa gave no history of bleeding. Hemorrhage from a Meckel's diverticulum occurs most frequently in infancy.

Various theories have been advanced to explain the presence of gastric mucosa in a Meckel's diverticulum. These are ably discussed by Goetsch,[7] Curd,[8] Edwards,[9] and Greenblatt.[10] The two chief theories, as described by Curd, are that it represents displaced fragments engrafted in the vitelline duct or, more probably, that the primitive endoderm has pluripotential powers and develops this type of mucosa. It may develop any type of digestive gland if stimulated locally, in some undetermined manner. This stimulus may be irritation, infection, lack of bile, retarded growth at different levels of the intestinal tract, etc. He found gastric mucosa in three of eighteen cases which he reported.

There is considerable speculation regarding the etiology of the giant diverticula. Edwards[9] presents a giant diverticulum 28.5 inches long arising in the mesentery a few inches from the ileocecal valve. Eighteen inches from the valve in the same case there was a typical Meckel's diverticulum. He feels that the giant diverticula are in no way related to a Meckel's but are in all probability true reduplications of the bowel. They represent attempts at the formation of a twin, this having come at a late stage of development, the reduplication being limited to one segment of bowel. Edwards reports many interesting reduplications of the bowel as found in the Royal College of Surgeons and St. Bartholomew's museums. Moll,[4] Yates,[11] Abt and Strauss[12] and Goldstein and Cragg[13] report giant diverticula. The giant diverticulum described by Edwards[9] and the one in this series both showed a mucosa made up of two parts, a superficial layer resembling intestinal mucosa and a deeper layer resembling gastric mucosa. He suggests that the diverticula contain acid juice but this was not true in our case.

Foreign bodies in a Meckel's diverticulum are unique findings. Abt and Strauss[12] refer to multiple concretions in one of their cases and this occurred in one of this series. An analysis of the concretions, which resembled gallstones, revealed that they were made up of dried intestinal contents. A bullet was present in one case. This must have entered the bowel and then passed along it until it lodged in the diverticulum. Even more interesting is the fact that it frequently presented in the right scrotal hernia and was palpated there. Webster[14] reports a case of an inflamed Meckel's diverticulum in a left hernia.

There seems to be a consensus of opinion that a patient presenting an unexplained anemia associated with blood in the stools, in the presence of normal stomach and colon by proctoscopic and *x*-ray examination, should be strongly suspected of having an ulcer of Meckel's diverticulum.

Acute inflammation of the diverticulum was present in only two instances and edema in a third. This would indicate that inflammatory processes are of less frequent occurrence than some of the other complications involving a Meckel's diverticulum. When patients come to operation with a diagnosis of appendicitis and the findings in the appendix are insufficient to account for the picture, a search should be made for a Meckel's diverticulum. This should consist in the examination of 3 or 4 feet of the terminal ileum.

Five methods of treatment were used by the seven surgeons. The usual and simplest method was to excise the diverticulum in the same manner as an appendix. This is very satisfactory in a majority of cases. The bowel was resected only when its blood supply was interfered with or the diverticulum was so large that an anastomosis seemed of less danger than the excision.

Inversion of the diverticulum is an easy method of treatment and it seemed to be a life-saving measure in one case. The possi-

bility of the inverted mass initiating an intussusception must be kept in mind. No other satisfactory method appeared possible in the patient in which it was used. Harbin,[15] in thirteen cases, inverted two and resected eleven. The exteriorization of the diverticulum was carried out in the strangulated hernia since no extensive operation could be performed.

CONCLUSIONS

1. Twenty-five Meckel's diverticula have been observed during the process of approximately 12,000 intraperitoneal operations, an incidence of 0.21 per cent.

2. Meckel's diverticula have been observed at the extremes of life. In this series one occurred at the age of 14 weeks and another at 77 years.

3. Twelve of the twenty-five diverticula, or 48 per cent, gave rise to symptoms which indicated their removal.

4. Meckel's diverticula should be removed in all cases where this procedure does not add appreciably to the risk of the operation.

5. Meckel's diverticulum is an important factor in the production of intestinal obstruction.

6. The preoperative diagnosis of Meckel's diverticulitis is difficult unless there are complications to suggest its presence. Vague abdominal pain associated with anemia and the passage of blood per rectum, usually occurring in childhood, is suggestive of the diagnosis of ulcer of Meckel's diverticulum.

7. Excision of the diverticulum with the same technique as used with the appendix is the usual method of choice. Resection of the bowel and inversion should be carried out only for special indications.

8. A search should always be made for a Meckel's diverticulum if the lesion at operation is insufficient to account for the preoperative symptoms and findings.

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A TECHNIQUE FOR ANAL REPAIR

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THE fragility and complexity of the anorectal tissues as demonstrated by recent anatomic studies[1,2] leave the important inference for the surgeon that operative trauma should be minimized. And because there is a direct ratio between the degree of such trauma and the degree of "working exposure" required for the proper performance of common rectal operations, it follows that the patient's welfare will be best served when such exposure is established at the minimum which is consistent with good surgery.[3]

Although the use of an operative speculum (not a bivalve type) is accompanied by the minimum of traction trauma, it is of value only to the surgeon who can adapt his procedure to the space afforded by the instrument. Uncontrolled bleeding, a hindrance to any operation, is a greater obstacle when dealing with tissues within the confines of a speculum. Direct vision is obscured and guiding landmarks lost. Further surgical progress is impeded when the operative field is obstructed with hemostatic forceps.

Aside from the difficulties that arise from impaired visibility, the actual manipulative technique or instrumentation becomes a definite problem if one is to accomplish his surgical aim in a field of restricted exposure.

These difficulties, however, do not constitute valid reasons for discarding this method in favor of those that are accompanied by traction trauma. Here, technique should be reasonably adapted to anatomic topography as it is in other branches of orificial surgery. Certainly, the ultimate advantages for both patient and surgeon justify a similar adaptation in rectal surgery.

Moderate experience in dealing with anorectal tissues in situ will usually yield to the operator many helpful ideas which facilitate this type of surgical procedure. In hemorrhoidectomy, for example, difficulties in regard to visibility and instrumentation may be prevented by consideration of the surgical anatomy in relation to the pathologic area. By instituting a progression of "surgical steps" which conforms to the anatomic arrangement of the anorectal tissues, the procedure may be simplified.

Since bleeding may be a troublesome factor, it is logical to direct the surgical approach and establish hemostasis at the highest level in the surgical field from whence bleeding emanates. This is the site of the deep branch of the inferior hemorrhoidal artery which enters the submucosal space of the canal above the profundus division of the external sphincter.[4] The level lies midway between two easily palpated landmarks: the intermuscular groove and the puborectalis muscle which separates the rectal ampulla from the anal canal. Hence, a preliminary transverse incision is made at this level which demarcates the underlying pathological area to be excised. (Fig. 1A.) With hemostasis established (by lightly drawing the curtain of the overlying membrane against the bleeding wall) the underlying row of infected crypts and hemorrhoidal membrane is accessible for dissection. (Fig. 1B.)

Provisions for the subsequent coverage of operative raw space are made when removing this pathologic tissue. By first bisecting the hemorrhoidal mass underlying the preliminary transverse incision, flaps are created from whose posterior surfaces the varicosed vascular plexus may be excised. (Fig. 1C.) After surplus tissue is trimmed from these flaps, they may be used to cover the operative raw space. (Fig. 1D.)

Adequate drainage of this infected field may be accomplished by the manner in which the overlying cut edge of the membrane is united to the subjacent tissue. (Fig. 1E and F.) Suturing this edge to the underlying sphincter in such a manner as to leave an intervening space will establish sufficient drainage to prevent postoperative deformity and to promote smooth healing.

Instrumentation is facilitated by selecting instruments whose size and shape conform to the restricted operative field. The preliminary transverse incision can be made most accurately with angulated

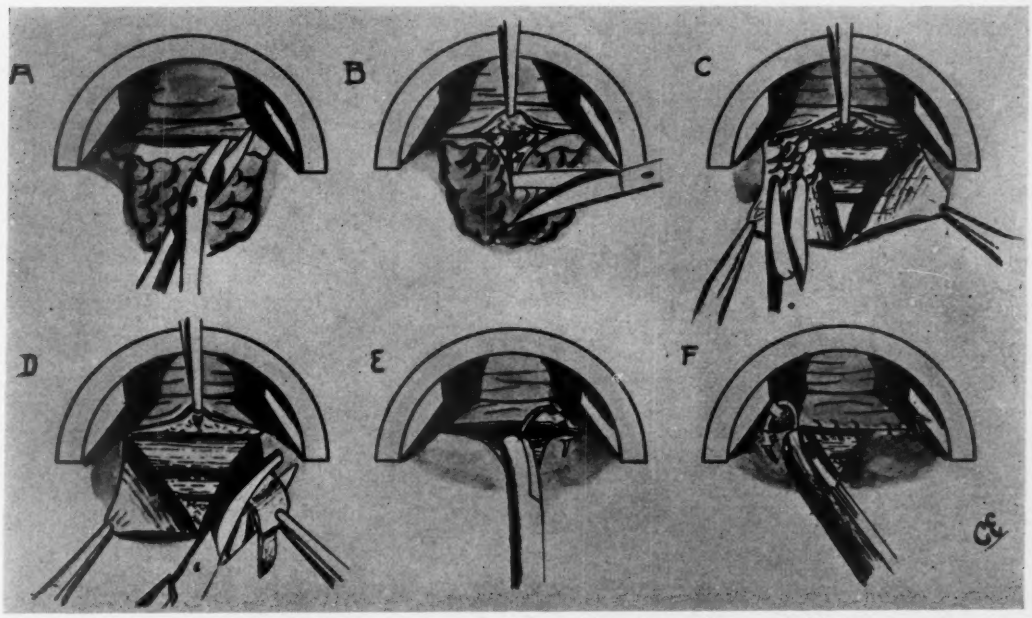

Fig. 1. A, transverse incision above the dentate line. B, bisection of hemorrhoidal mass. C, excision of hemorrhoidal varicosities. D, removal of redundant membrane. E, beginning of closure. F, half Lembert sutures with drainage defect.

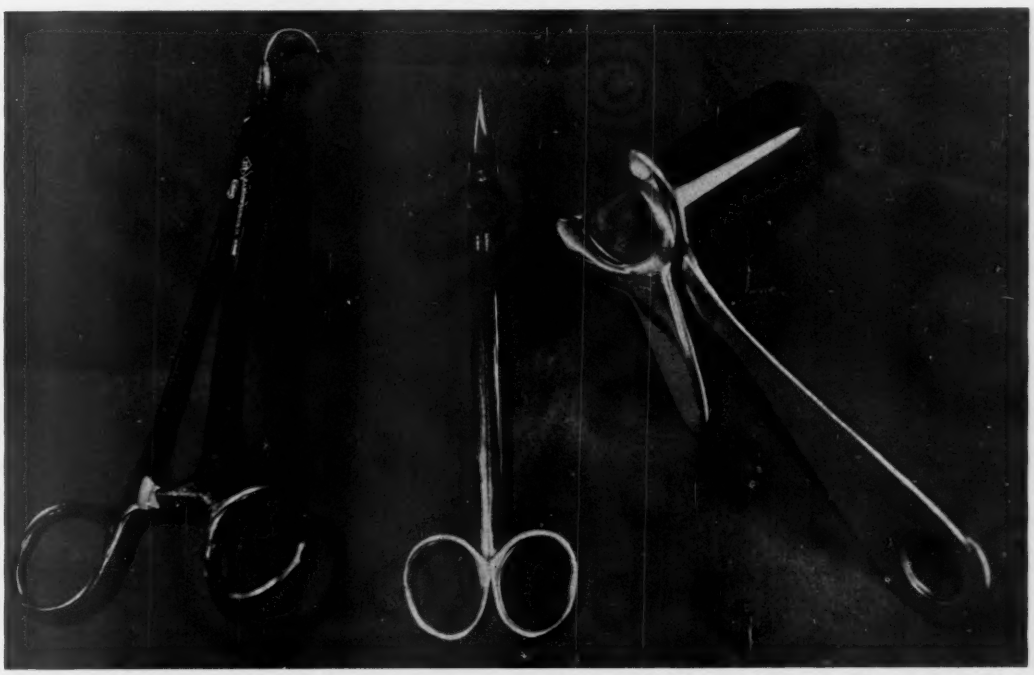

Fig. 2. Showing curved needle holder, angulated scissors, and operative speculum containing revolving obturator.

scissors. Also, a curved needle holder that grasps the needle at its tip, holding it at the proper angle, is of advantage. Finally, a nontraumatic speculum that affords 180 degree exposure has been found adequate. (Fig. 2.)

SUMMARY

With the conviction established that better operative results, with less pain and disability for the patient, can be obtained in rectal surgery by minimizing traction trauma, suggestions are made to facilitate the use of a restricted field of surgical exposure.[5]

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Varicose veins of the vulva rarely require surgical treatment. When extremely large, however, they may be excised, preferably with the patient under general anesthesia and with the usual strict precaution in regard to hemostasis which this region requires.
From—"Minor Surgery" by Christopher (Saunders).

ARTERIOSCLEROTIC GANGRENE—A MAJOR CLINICAL PROBLEM*

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ARTERIOSCLEROTIC gangrene constitutes one of the most serious of clinical problems. When one considers that most of these patients come to major amputation, and that the mortality of major amputation for gangrene, as reported from various clinics, ranges from 13 to 65 per cent, the seriousness of the disease is apparent.

The higher mortality rates occur when: (1) patients present themselves for treatment in a moribund condition; (2) insufficient attention is paid to details concerned with the medical care of these patients, their preoperative preparation, and the operation itself; (3) when treatment designed to effect a local separation of the gangrenous part is employed in unsuitable cases, or when it is extended beyond the point where it is safe. The last point requires considerable elaboration because in recent years there has been an attempt in certain quarters to create the impression that arteriosclerotic gangrene is usually amenable to various forms of so-called "conservative" peripheral vascular therapy. This has proved to be unwarranted and dangerous. A little reflection on the basic pathology of the disease enables one to understand why.

Arteriosclerotic gangrene involves chiefly the toes, which receive their blood supply through end arteries. Should these vessels thrombose, there is little chance for the rapid development of an effective collateral circulation; and the ischemia often progresses to frank gangrene. Once gangrene occurs, nature makes an effort to separate and slough off the dead part. Gangrenous toes are separated by an inflammatory process involving all the forces of destruction and repair. Since the plane of separation must pass through fascia, tendon, and bone, as well as skin and subcutaneous tissue, a good blood supply to the zone of demarcation is essential if separation is to take place. This is usually not present. If it is, the intense inflammatory reaction in the adjacent tissue may produce further arterial thrombosis with consequent spread of the gangrenous process. With a spread-

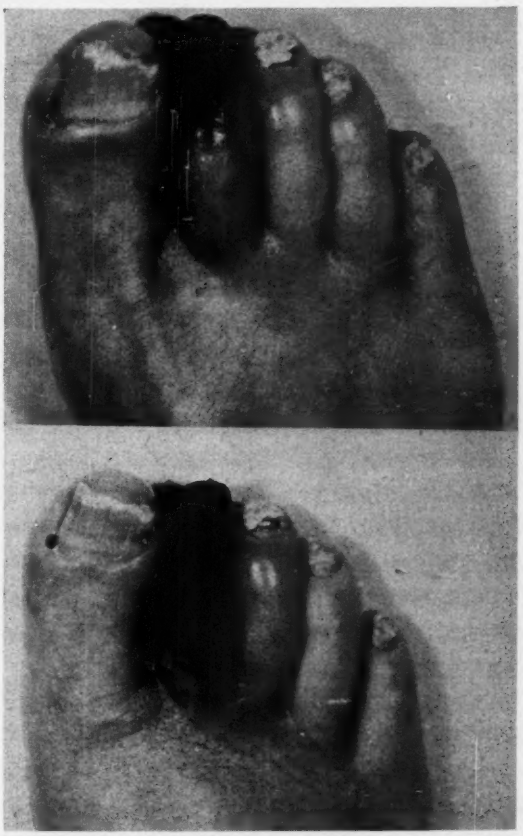

FIG. 1. Diabetic with advanced arteriosclerosis. No pulsation palpable in pedal arteries. Oscillometric reading lower leg—zero. Upper photograph taken on admission; lower one week later. Supracondylar amputation advised but refused. Patient expired from sepsis.

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ing gangrene, there is the danger of absorption of toxic products. Such an absorption may affect seriously an associated diabetes slim that the life of the patient is jeopardized to attain an unusual and spectacular result.

FIG. 2. Diabetic with minimal arteriosclerosis. Good pulsation palpable in pedal arteries. Oscillometer registered good pulsation in lower leg. Gangrene of both great toes followed burns from hot water bottle. Gangrenous tissue demarcated, separated, and residual ulcers healed.

or a diseased myocardium or renal parenchyma. (Fig. 1.) Finally, there is the specter of infection. Every patient with arteriosclerotic gangrene is an excellent candidate for gas bacillus infection, tetanus, and septicemia; and because of the poor blood supply to the involved extremity, the progression of the infection may be extremely rapid and fatal.

Considered in the light of its pathology, arteriosclerotic gangrene can be looked upon as a fatal disease, and major amputation as a life saving measure.

The question arises when, if ever, is conservative treatment indicated and safe. It is very difficult to generalize. Each case presents its own particular problems. We consider it safe to attempt tentative conservative treatment designed to effect a local separation of the gangrenous part in any case of arteriosclerotic gangrene when:

1. The gangrenous process is localized to a single toe. There have, of course, been reports of conservative treatment successfully employed in instances of more extensive gangrene than this. However, the chances of successfully terminating conservative treatment in instances of more extensive arteriosclerotic gangrene are so

2. The gangrenous process is well demarcated with no tendency to spread.

3. There is no constitutional evidence of toxic absorption.

4. There is no cellulitis, osteomyelitis, or lymphangitis.

In the above type of case there is little pain, no fever, no loss of appetite or weight, no persistent tachycardia, and no retention of nitrogenous waste products. Any associated diabetes will be easily controlled. While diabetes, per se, is no contraindication to conservative treatment in this type of case, it has been my experience that failure is to be expected unless an adequate circulation is present in the uninvolved portion of the foot. (Fig. 2.) For interesting and informative statistics on this phase of the subject, the reader is referred to McKittrick.[1]

Our method of handling these cases conservatively is very simple. Every patient receives a prophylactic injection of gas and tetanus bacillus antitoxin on admission. We do not use any of the various types of passive vascular apparatus until complete separation of the gangrenous part has occurred. We have several times seen a rapid spread of gangrene in apparently favorable

cases follow the use of passive vascular machinery. Complete bed rest is enforced. The leg is kept at a lower level than the heart. Many of these patients feel better if they are allowed to sit up in bed. A cradle without lights is used to protect the foot, and the gangrenous toe is kept scrupulously clean and protected with sterile dressings saturated with azochloramid,[2] or a suspension of zinc peroxide in sterile distilled water.[3] The leg and foot are swathed in cotton to conserve body heat. Unless contraindicated, 100 c.c. of 5 per cent saline are given intravenously every other day, and whiskey is used as a vasodilating drug. My experience with enzyme-free pancreatic extract has been limited. However, in view of recent enthusiastic reports,[4] it deserves trial. My experience with peripheral nerve crush, periarterial sympathectomy, and lumbar sympathectomy in the treatment of arteriosclerotic gangrene has not been encouraging. When the gangrenous tissue has been converted into a loose sloughing mass, and all reaction in the adjacent healthy tissue has subsided, and not until then, the gangrenous part is gently detached from the foot. A granulating ulcer remains. (Fig. 3.) At this stage, treatment with the intermittent venous occlusion apparatus is very useful to assist in healing the residual ulcer.

FIG. 3. Arteriosclerosis not complicated by diabetes. No pulsation palpable in pedal arteries. Oscillometric reading lower leg—zero. Shows ulcer remaining after separation of gangrenous toe.

FIG. 4. Arteriosclerosis not complicated by diabetes. No pulsation palpable in pedal arteries. Oscillometric reading lower leg—zero A case of infected subungual ulceration of great toe with gangrene of nail bed and adjacent tissue. Shows end result following treatment as outlined in text.

At the first sign of a spread of the gangrenous process, of the onset of infection, or of toxic absorption, conservative treatment is terminated and major amputation is performed. Here, as in conservative treatment, the coöperation of an internist skilled in the handling of diabetic, cardiac and renal problems is desirable. These patients have a much better chance of surviving their operation if pains are taken preoperatively to replenish their depleted body fluids, to build up their nutrition, and to correct associated medical difficulties. The operation of choice is the well known circular amputation through the lower

third of the thigh with primary closure, using a low spinal anesthesia of no more than 50 mg. of novocaine. In the event of a cellulitis of the foot or a lymphangitis, a simple guillotine through the upper third of the leg with the stump left open is rapidly performed under nitrous oxide anesthesia. The open stump is treated with zinc peroxide using the technique described by Meleney.[3] Sulfanilamide, deep *x*-ray therapy for gas bacillus infection,[5] and small blood transfusions are used where indicated. At a later date, when all risk of infection of a primarily closed stump has disappeared, necessary stump revisions or reamputation at a higher level may be performed. A thigh amputation with primary closure in the presence of a cellulitis of the foot or a lymphangitis carries a grave risk of a badly infected stump. These elderly patients do not tolerate an infected amputation stump any better than they do an infected foot, and often succumb. Infected amputation stumps constitute one of the leading causes of mortality. (According to J. Ross Veal,[6] the danger in an infected amputation stump is that of septic pulmonary embolism. He has effected a large reduction in his mortality by the simple expedient of ligating the femoral vein just distal to the entrance of the saphenous vein at the time the amputation is performed.)

A type of gangrene occasionally seen in ischemic feet is in a class by itself. It involves the soft parts at the tip of, usually, the great toe, and is initiated by a neglected infection starting beneath the nail. Gentle removal of the "floating" portion of the nail to allow drainage of the infection, equally gentle removal of a separating gangrenous slough, and the application of mild antiseptics often yield gratifying results. Here again, the intermittent venous occlusion apparatus is useful to assist in healing the residual granulating ulcer. (Fig. 4.)

True diabetic, or the so-called "wet" type of gangrene, is not essentially a problem of ischemia, and need not concern us here. It is infectious in origin, and has to do with the question of low local tissue immunity peculiar to diabetes. Its treatment and prognosis depend on the nature of the infection and the severity of the diabetes. Inadequate blood supply, of course, renders the condition more serious.

SUMMARY

1. Arteriosclerotic gangrene is a serious clinical problem with a high mortality.

2. One of the causes of this mortality is the failure to perform major amputation as soon as it is indicated.

3. The indications for, and the technique of, conservative treatment and surgical interference are discussed.

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ADRENAL GLANDS AND MALIGNANCY*

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FIFTEEN years ago, a general opinion prevailed among the carcinologists that the endocrine system had no direct or even indirect connection with the evolution of malignancy. At that time, only a few, and most notably Dr. Leo Loeb, suspected that some of the hormones might have carcinogenic properties. During the intervening period, however, considerable material has accumulated strongly indicating that the endocrine system may be directly or indirectly involved in the process of neoplastic formation.

Lacassagne and Loeb, and after them numerous other investigators, demonstrated the carcinogenic activity of estrin. In certain strains of mice in whom mammary cancer occurs spontaneously in only a very small proportion of the females (2 to 3 per cent), the normal rate was increased by estrin injections to 60 per cent in both sexes.[1] An important observation has been recently reported by Geschickter, who was able to obtain, by injection of estrogenic substances, a large number of mammary adenocarcinomas in both male and female rats of a strain in which since 1934 no spontaneous cancer had been registered in a colony of 2000 animals.[2]

Corner suggested that the ovarian hormones act on the mammary gland through the intermediary of the anterior pituitary. Nelson[3] injected estrin and noticed a diminution in the number of basophiles in the pituitary and the disappearance of castration cells in the previously castrated animals of both sexes. He obtained a greater reaction in the female than in the male rats. Collip and his co-workers[4] found that administration of large doses of estrin to female rats led to a hypertrophy of the anterior lobe within a few days—an effect which was less marked in males and in castrates. Other observers[5] found that estrin increased the weight of the pituitary in males, while McEuen, Selye and Collip[6] reported that adenoma formation sometimes occurred. According to Wolfe and co-workers,[7,8] the change caused by estrin treatment in the anterior lobe of the rat's pituitary is manifested in a degranulation of the eosinophile and basophile cells and in an increase in size and number of the chromophobe cells.

B. Zondek[9] observed that following prolonged administration of estrin the function of the anterior pituitary is inhibited. The gland becomes enlarged and increased in weight in males, while in females pituitary tumors may be observed. Cramer and Horning[10] reported that a prolonged application of estrin to mice resulted in functional inactivity, producing a condition resembling that following hypophysectomy; a hyperplasia of the anterior lobe was observed with noticeable diminution of chromophiles and increase of chromophobe cells. According to Burrow,[11] this hyperplasia occasioned by estrin could be observed in males only on rare occasions.

Halpern and D'Amour[12] observed that the administration of estrin causes an increase of 100 to 200 per cent in the weight of the hypophysis, produces hypertrophy and hyperplasia of the chromophobe cells, with increased mitosis. These effects were interpreted as an indication that estrin causes a release of gonadotropic hormones, and that when the secreting capacity of the gland is excelled by demand, compensating hyperplasia of the primary cells results.

In general, a greater reaction takes place in the females than in the males. Thus, in large doses, hypertrophy of the anterior lobe is noted within a few days in females. Following prolonged administration of

* Report to the Annual Meeting of the North American Radiological Society, November, 1938.

estrin there appears an enlargement of the pituitary with production of adenomata, an inhibition of the activity of the anterior lobe and of the growth and sex hormones.

Karlefors[13] in 1920 reported that in cancer (in man) the chromophobe cells of the pituitary were increased in size. Rössler[14] observed an increase in the eosinophiles in case of chorionepithelioma. Guyer and Claus[15] described changes in the anterior pituitary in rats who were bearers of Flexner carcinoma. The anterior pituitary was found to resemble that of castrated animals, with an increase in basophiles and with hyperactivity. These investigators concluded that changes are brought about by the influence of the growing tumor on the gland. When carcinoma is transplanted to the uterus, there is an increase in acidophile cells in the pituitary, resembling the phenomenon observed following estrin injections. Wyeth[16] found an increase in the eosinophile cells in the presence of human cancer. The gland is also heavier than normal (weight increase of 0.60 to 1.80 Gm.). Engel[17] recorded loose structure of pituitary in the majority of tumor mice. Mendeleef[18] concludes that pituitary hormonal activity increases during the evolution of a tumor and that the hypophyseal growth hormone is responsible for the continued proliferation of a neoplasm once this has been initiated. Lacassagne and Nyka[19] observed that after the hypophysis has been destroyed, rabbits appeared to be less susceptible to carcinogenic action of both tar and benzopyrine, as well as to inoculation with Shope papilloma. Samuels and Ball[20] confirmed this observation. They found that the pituitary is a factor in tumor growth, since hypophysectomy retards the growth rate of Walker carcinoma in rats and also slows the rate of growth of subcutaneous tumor induced by debenzanthracene.

Summarizing these observations we may say that there seems to be a definite increase of basophiles, as well as of pituitary hormone activity in malignancy, the reverse of which is observed under estrin therapy. Gonadectomy also leads to hypertrophy of the pituitary, with an increase of basophile cells and an increase in the gonadotropic hormones.

These observations are receiving particularly interesting interpretation in the light of the recently published work by Loeb and Kirtz.[21] The transplantation of anterior lobes of the hypophysis causes a marked development and secretory activity in the mammary gland tissue, both in strains with a high and with a low incidence of spontaneous mammary gland carcinoma. These transplants also cause a considerable increase in the cancer rate. The authors point out that the mechanism depends upon the coöperation of functioning ovaries, transplantation being entirely ineffective in ovariectomized mice. There is a possibility that the suppression of the luteal hormonal activity and the follicular hormonal overactivity is a contributing factor directly promoting growth processes in the mammary glands. In a number of mice which had received anterior lobe transplants there occurred, in addition to the mammary gland changes, precancerous proliferations in the vaginal-cervical tract.

In his previous work Loeb,[22,23] has established that ovariectomy causes a marked reduction in the incidence of mammary cancer. If in mice belonging to strains with a high incidence (almost 100 per cent) of mammary adenocarcinoma, the ovaries are extirpated at the age of 3 or 4 months, the cancer incidence falls to zero. These findings, confirmed by other workers (Cori, Laccasagne) are now interpreted as indicating that excess of follicular hormone, acting on the mammary gland, causes the transformation of normal mammary gland tissue into cancerous tissue.

Glandular imbalance, observed in mammary cancer, is not limited to the anterior pituitary and the ovaries. In 1929, Sokoloff[24] found that the adrenals of chickens who were bearers of Rous sarcoma, increased to from three to four times their normal weight. Some increase in the weight of adrenals was found in Flexner carcinoma rats and in sarcoma #180 mice. In 1934,

Tamura[25] confirmed these observations. More recently, Sure, Theis and Harrelson[26] found that in Walker carcinosarcoma whether spontaneously in the females or in response to estrin in the males. This phenomenon was confirmed subsequently

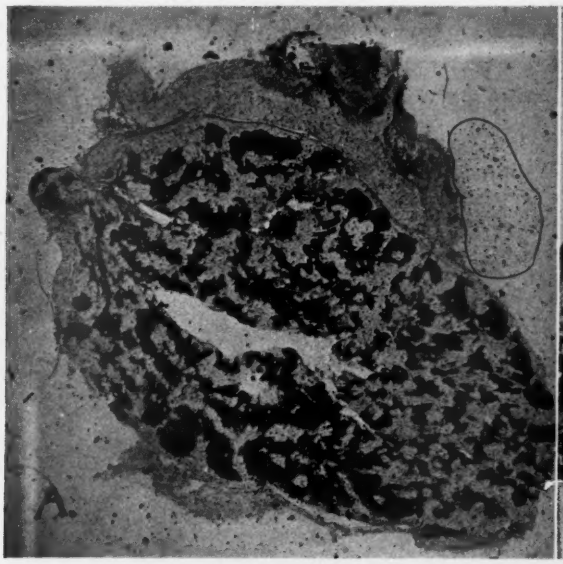

Fig. 1. Adrenal cortex of normal chicken (Sudan III).

Fig. 2. Adrenal cortex of Rous sarcoma chicken showing fatty degeneration (Sudan III).

the weight of the adrenal is almost doubled.

Okamotto[27] found that Kato rabbit sarcoma elicits proliferative changes in the adrenal cortex of rabbits and according to Oike[28] in tar cancer the adrenals of rabbits are greatly enlarged.

McEuen and Selye[29] and Sala and Stein[30] described a leucocytic infiltration of the adrenals of tumor-bearing rats, particularly in the zona fasciculata. This was thought to be due to the necrosis of adrenals.

Cramer and Horning[31] observed a brown degeneration in the adrenals of mice of mixed strains which had been painted with estrin. Isolated lipoid-containing cells in the zona reticularis were enlarged and their contents were transformed into a brown material. Degeneration became more extensive during prolonged administration of estrogenic substances. Although brown degeneration did not occur in normal mice, it occurred spontaneously in male and female mice where there was a high incidence of mammary carcinoma among the females. The important point, according to these authors, is that these adrenal lesions fully developed *before* mammary cancer occurred, by Lacassagne and Raynaud,[32] Burrows,[33] and Dobrovolskaia-Zavadskaia.[34–35] In a recent publication, Cramer and Horning[36] reported that in the two high cancer strains the brown degeneration began very early and after six months was present in all mice. Males and females of equal ages were found to be about equally affected. The brown degeneration affected not only the cortex but the medullary cells of the gland as well. They believe that "an impairment of the functional activity of the adrenal medulla favors the action of the ovarian estrogens on the mamma." Experiments on adrenalectomized mice had led them to the conclusion that "the hormonal functions of the adrenal cortex are synergic with the estrogenic functions of the ovary."

Ball and Samuels[37] found that the adrenals are enlarged in tumor-bearing rats. But in hypophysectomized rats bearing Walker carcinoma this hypertrophy of the adrenals was absent and, as is usual in hypophysectomized animals, atrophy of the adrenals was present. The authors conclude that the reaction of the adrenals to tumor growth is not a direct one but a reflection

of modification in pituitary hormonal activity in case of malignancy.

Reviewing these observations leads to an

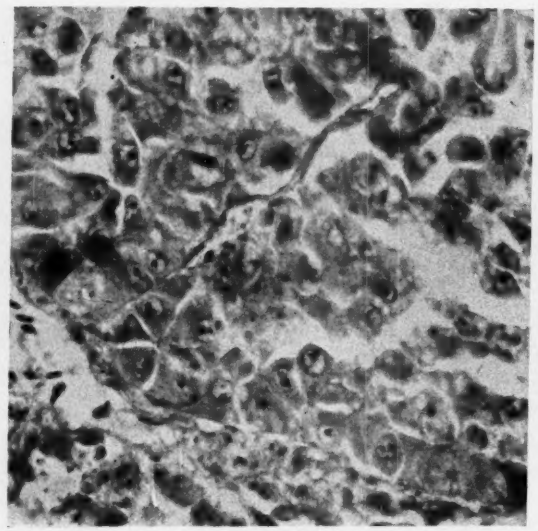

Fig. 3. Adrenal cortex of Rous sarcoma showing vacuolation of spongiocytes.

indication that in malignancy, as well as under estrin application, there occur histologic changes in the adrenals which seem to have a close interrelation with the reaction of the anterior pituitary.

Our study on this subject has been conducted for over a period of years, embracing extensive material. We have examined the adrenals of chickens, bearing Rous sarcoma; of rats, bearing Flexner carcinoma, sarcoma #39 and #10; and of mice, bearers of sarcoma #180.*

Table 1
ADRENAL WEIGHT IN CHICKENS

Types	No.	Average Weight, Mg.
Normal	5	100.8
Small tumor	5	136.0
Medium tumor	10	169.0
Large tumor	10	332.0

The most striking changes were observed in the adrenals of Rous sarcoma chickens. With the progress and growth of tumor the weight of adrenals steadily increased, reaching as high as four times the normal weight. (Table 1.)

The adrenal hypertrophy in tumor-bearing chickens affects chiefly the cortical part of the gland. (Figs. 1 and 2.) The cortical cells in malignancy are increased in volume, lose their typical structure and become filled with fat. The spongiocytes are in a state of vacuolization (Fig. 3), with picnotic nucleus and modified cellular structure.

The adrenals of tumor mice and rats show less pronounced increase in weight than in chickens. The average increase is about 80 to 90 per cent of the normal rate. Here there are two distinct stages of glandular reaction: cellular *hyperactivity* in the beginning of tumor growth, and *hypoactivity* and pathologic lesions in advanced malignancy.

In the adrenal of the female mouse the medulla and cortex are not separated by a defined band of connective tissue, as they are in the male. The juxtamedullary zone of reticular cells almost completely disappears when the males reach the age of two months but in the females it continues to growth until puberty, but it is here that the first cellular reaction to tumor growth is observed. The reticular cells, normally free of pigmentation, show the first signs of granulation and mitochondrial activity. The number of Altmann granules, which are considered as representing prelipoids, are also increased. Gradually, with growth of the tumor, the fatty degeneration is more pronounced, the Altmann granules disappear almost completely and the spongiocytes are filled with fat.

Soon after the transplantation of a tumor and with the first sign of the tumor growth there is an increase in the number of large reticulo-endothelial cells, particularly in the zone outside the medulla. With the progress of malignancy these cells disappear almost completely from the zona reticularis and only a few plasma cells are found. Intensive vacuolization of cytoplasm around the nucleus in plasma cells indicates a specific regressive process (Stoerk).

* A large part of this work was done by one of us (B. S.) in Dr. Leo Loeb's Laboratory at Washington University Medical School, St. Louis.

Gradually, the zone fasciculata also shows a sign of degeneration. (Figs. 4 and 5.)

Cortical Extract. In 1930, Arloing, Josserand and Charchon[38] found that adrenal cortex extract had inhibiting and even curative effects in malignancy, but a number of other authors[39-41] could not achieve these results. More recently, Beard[42] using concentrated cortical extract and supracorsin was able to obtain some inhibiting effect on the growth of Walker carcinosarcoma. We have found that Wilson cortical extract had no effect on sarcoma #180. Large doses of the same extract produced a slight inhibiting effect on Flexner rat carcinoma.

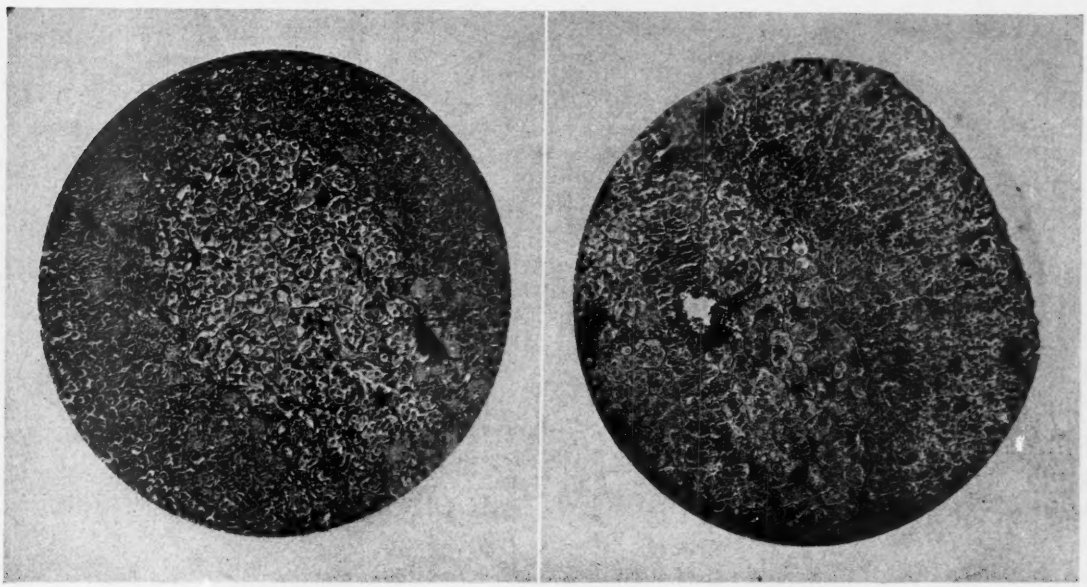

Fig. 4. Fig. 5.

Figs. 4 and 5. Adrenal cortex of mouse with sarcoma.

Adrenalectomy. Rogoff and others have demonstrated that bilateral adrenalectomy has a pronounced inhibiting effect on the growth of transplanted tumor. Tamura,[41] however, found only slight inhibition from destruction of both adrenal glands.

According to our experiments on twenty rats, unilateral adrenalectomy had no effect on the growth of sarcoma #39. The operation was performed at the same time when the graft of tumor was made. Bilateral adrenalectomy had a definitely inhibiting effect on the growth of Flexner carcinoma and sarcoma 39, if the graft followed the adrenalectomy.

Bilateral adrenalectomy was performed on thirty rats. Ten days after the operation, tumors were grafted (twelve rats with Flexner carcinoma, eighteen rats with sarcoma #39). The animals were maintained on small amounts of suprarenal extract, injected subcutaneously. The adrenalectomized animals were rather apathetic and lacking in their usual vitality. Two animals died, probably from large tumors (sarcoma 39), and two others from infection. The rest seemed to show a strong resistance towards grafts. In the control animals, two rats out of eighteen showed regression of tumors, the remaining sixteen dying from malignancy.

Similar results have been obtained with Flexner carcinoma. Only in two instances out of twelve did the adrenalectomized animals die from malignancy.

In other series of experiments, in which the graft preceded adrenalectomy, the inhibition of the tumor growth was much less pronounced.

Considering the fact that bilateral adrenalectomy reflects considerably on the general condition of animals, the inhibiting effect of destruction of adrenals on tumor growth should be minimized. We know that the general well-being of the animal is an essential factor for successful transplanta-

tion of cancer. Nevertheless, adrenalectomy seems to have some inhibiting effect on tumor growth.

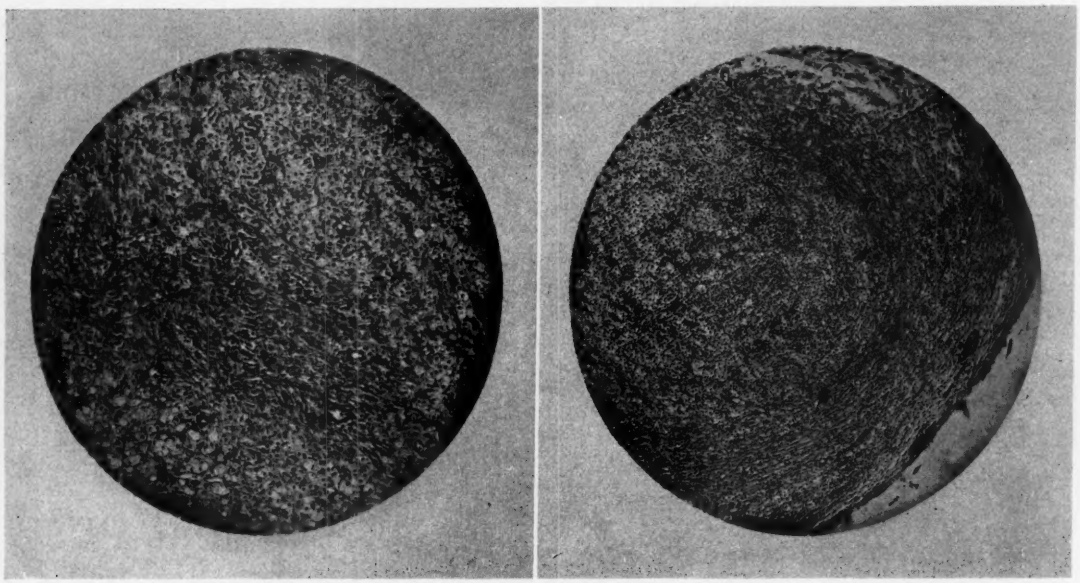

Fig. 6. Fig. 7.

Figs. 6 and 7. Transplanted adrenal cortex showing initial necrosis (mouse).

Adrenal Grafts. Adrenals extirpated from brothers or sisters were transplanted to rats which had already been inoculated

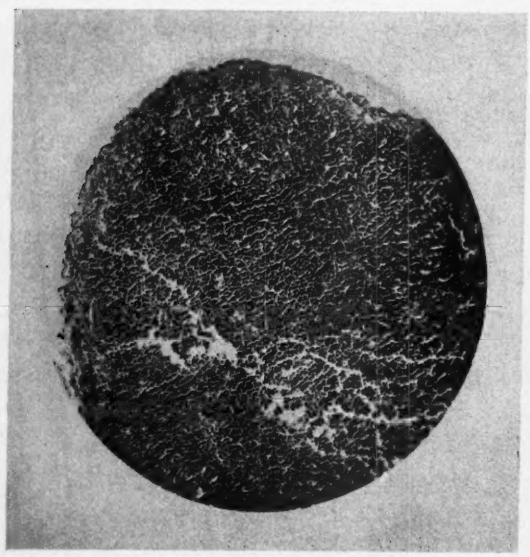

Fig. 8. Initial necrosis.

with sarcoma #39 (thirty rats) or Flexner carcinoma (twenty rats). The adrenal gland was transplanted either to ovaries or intracutaneously.

As a rule, degeneration of the transplanted gland followed within forty-eight to seventy-two hours after the operation. Initial necrosis manifested itself in the center of the gland, then gradually extended peripherally. (Figs. 6 and 7.) Three days after grafting, the adrenal gland, except for the capsule and the exterior portion of the zona glomerulosa, was destroyed.

At about this stage the first signs of regeneration of the cortical tissue were noticed, but this regenerative activity of the cortical tissue was very small and the amount of restored cortex was rather insignificant.

We could not observe any noticeable effect of the grafting on the growth of the tumor. In three cases there was some inhibition of tumor growth, but this was only temporary.

Studying the regeneration of cortical tissue of grafting adrenals, we found considerable difference in regenerative power, depending on the progress of malignancy. In those cases where the animals had small incipient tumors, the adrenal transplant showed much less regenerative activity. In cases where the adrenals were grafted in animals with large tumors, the regenerative process was much more extended. The organism of animals affected with advanced malignancy must have some physiologic

need for the biologic principle elaborated by the adrenal gland. The function of the adrenal cortex seems to be in a suppressed stage in cases of advanced malignancy.

Recently Wyman and tum Suden[43] concluded from a series of their studies on adrenal grafts that survival of a graft depended on the presence of the pituitary adrenotropic principle. They expressed the belief that this was used in the growth and maintenance of the normal adrenals, and thus adrenal grafts without available adrenotropic principle failed to survive. Their theory finds confirmation in the recent work of Ingle.[44]

DISCUSSION

Regaud pointed out that in all probability a state of hyperactivity of some glands precedes the appearance of malignancy. Yet the nature and character of this glandular dysfunction has not been clearly defined. However, it is a well established fact that prolonged application of estrin inhibits the activity of anterior pituitary, including the production of the growth and sex hormones. Should we therefore consider estrin over-production as a potential precancerous state, since estrin has definite carcinogenic properties?*

Some evidence in support of this theory was brought out in the recent work of Leo Loeb, who found that anterior pituitary transplants increased the incidence of mammary gland carcinoma in inbred strains of mice. However, the transplants of hypophysis were completely ineffective when the ovariectomy preceded the transplantation. Thus, Loeb concludes, the growth-stimulating effect of anterior pituitary transplant on the mammary gland is not direct but through the ovaries, in all probability through the corpus luteum.

Corpus luteum seems to have an inhibiting effect on the growth properties of estrin. Thus we may visualize that an increase of anterior pituitary's activity may result in suppression of corpus luteum and deliberation of estrin-growth-stimulating factor.

These experimental data might be of considerable importance in cancer therapy, particularly in the case of malignancy of the mammary gland or uterus. They would suggest a sterilization in the earliest stage of malignancy as the most rational method of prevention of recurrence of growth.

The functional relations between the adrenal cortex and gonads are very close. The most striking fact in this relationship was brought out by Rogoff[46] who showed that pregnancy has a marked influence in dogs in prolonging the period of survival and maintaining good health after the removal of adrenals. The corpus luteum can presumably contribute something to make up for the loss of the adrenals. Goormachtich[47] recently suggested that the juxtamedullary cells of the adrenal and the luteal cells of the ovaries have something in common. Every change in the activity of the gonads reflects on the adrenal cortex, and vice versa. In B-avitaminosis, the adrenals may be enlarged from five to seven times, there may also be a striking atrophy of the gonads (in pigeon).

Thus the question arises, what rôle, if any, does the adrenal gland play in malignancy? Cramer found that the adrenal cortex of mice with a high incidence of mammary carcinoma has pathologic lesions which could not be observed in normal mice and which preceded the appearance of malignancy. Tamura confirmed the observation of one of us (Sokoloff) published some years ago, that the weight of the adrenal gland is increased in malignancy. A number of other authors have recently described various histologic changes in the adrenal cortex of cancerous rats and mice.

Analyzing our material on this subject, we found that there are at least two different cellular reactions of adrenal cortex to tumor growth. In the precancerous state

* Biologically, the suppression of growth hormone could be responsible for abnormal cellular multiplication. Suppression of the growth of cell increased the dynamic properties of its nucleus. According to Sokoloff,[45] the nucleocytoplasmic ratio of cancer cells of incipient growth is considerably increased.

there exists in the reticular zone of the adrenal cellular stimulation and glandular hyperactivity. In advanced malignancy, there is cellular degeneration and glandular hypofunction.

These experimental findings seem to be in accord with clinical observations on cancer patients. The feeling of well-being and even of unusual energy, which has often been noticed in patients with incipient cancer, is soon replaced by a feeling of extreme fatigue which often cannot be explained by anemia or toxemia, but may well be accounted for by the presence of lesions in the adrenal. We have, however, no proof that these adrenal lesions are contributing factors to the progress of malignancy. They may be nothing but a reflection of gonado-pituitary dysfunction. And even the fact that bilateral adrenalectomy considerably inhibits the growth of transplanted tumor does not necessarily prove that the adrenal plays an active rôle in cancer. Only one statement may be made safely at present: that in malignancy cortico-adrenal imbalance is present.

SUMMARY

The interrelation between tumor growth, the anterior pituitary, and the adrenal glands is discussed.

The histologic changes in the adrenals of chickens, rats, and mice, bearing tumor, have been studied. The weight of adrenal glands is increased in cancerous animals, particularly in chickens with Rous sarcoma.

Two stages of cellular reaction have been found in the adrenal cortex, hyperactivity of the zona reticularis, in incipient malignancy, and lipoid accumulation and fatty degeneration of the cortical part, similar to B-avitaminosis in large tumors. These two stages probably correspond to two different stages of glandular activity: hyperactivity of the adrenal cortex in the beginning of malignancy and hypofunction with the progress of tumor growth.

Unilateral adrenalectomy seems to have no effect on the growth of transplanted tumor. Bilateral adrenalectomy has a definite inhibiting effect upon it.

Grafting of adrenal tissue does not produce any noticeable effect on tumor growth.

The regenerative power of the cortical tissue is more pronounced in grafts in rats with large tumors than in non-cancerous rats. This suggests that the organism affected by cancer must have some physiologic need for the biologic principle elaborated by the adrenal gland.

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A STUDY OF SIXTY CASES OF TETANUS*

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THE mortality of tetanus today is just about what it was before specific antitoxin was developed. Graves and Hessart found the mortality in the pre-antitoxin era to be between 41 per cent and 84 per cent, or an average of 62.5 per cent.[4]

A recent study of 642 cases of tetanus by Huntington and his co-workers showed the total mortality to be 63 per cent.[3] These cases were collected from the Barnes Hospital, St. Louis Children's, Massachusetts General, Johns Hopkins, University of Pennsylvania, Presbyterian and Bellevue in New York, Boston Children's, and several others. Many other recent authors report mortality rates varying from 40 per cent to 72 per cent.

TABLE I

	No. of Cases	Deaths	Per Cent Mortality
Acute	32	23	71.8
Chronic	28	5	17.8
Combined	60	28	46.6

Acute—incubation period of ten days or less.
Chronic—incubation period of more than ten days.

A series of fifty-two cases of tetanus in the Vanderbilt University Hospital and eight cases in St. Thomas Hospital were studied, covering the past fourteen years. Two very doubtful cases and one case in which death probably resulted from other injuries and complications were excluded. The cases were arbitrarily divided into two almost equal groups according to the incubation period. Those patients whose symptoms appeared within ten days after the initial injury were classified as having early tetanus, while those with an incubation period of over ten days were termed late tetanus. The mortality of the early cases was 71.8 per cent, while the mortality of the late cases was 17.8 per cent. (Table I.) The mortality for the entire group was 46.6 per cent. There was almost an equal number of early and late cases.

There was no uniform method of treatment given all patients, as they were admitted on the pediatric, medical or surgical services of these two hospitals.

Puncture wounds of the extremities were the most frequent injuries. Nails, splinters, thorns, and rake prongs were the commonest agents. Lacerated wounds were the next most frequent. There were three cases in which a toe or finger was mashed, but only a contused wound sustained. Two of these patients died.

TABLE II

Year	No. of Cases	Deaths	Per Cent Mortality
1925–1931	24	11	45.8
1932–Oct., 1938	36	17	47.2

The mortality rate from year to year shows little change. The mortality rate in 1925 was 75 per cent and again in 1938, with an equal number of cases, 75 per cent. During the first seven-year period studied the rate was 45.8 per cent in a total of twenty-four cases. In the past seven years the mortality has been 47.2 per cent, with a total of thirty-six cases. (Table II.)

Three patients out of the sixty studied received prophylactic injections of at least 1,500 units of antitoxin within twenty-four hours of the initial injury. Two of these patients died and one recovered. It may be

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significant that all three of these patients had sustained fractures, two of them being compounded. There was soft tissue injury and wound conditions favored anaerobic organisms in these two cases.

Table III

Site of Injury	No. of Cases	Deaths	Per Cent Mortality
Face	3	2	66.6
Upper extremity	11	7	63.6
Umbilicus	3	2	66.6
Uterus	2	1	50
Lower extremity	41	16	39
Total	60	28	46.6

Klopp of the Episcopal Hospital in Philadelphia studied fifty-two cases of tetanus.[2] Of these five had received prophylactic antitoxin, but four of them died later of tetanus.

It is generally assumed that prophylactic antitoxin will entirely prevent tetanus, but 5 to 10 per cent of the patients with

Table IV

Age	No. of Cases	Deaths	Per Cent Mortality
0–5	15	7	60
6–15	25	9	36
16–55	17	7	41.1
56–70	3	3	100
Total	60	28	46.6

tetanus have had prophylactic antitoxin. The strongest evidence for prophylaxis is the low incidence of 0.117 per cent of tetanus in over two million wounded British soldiers who had received antitoxin.[4] The practice of debriding and irrigating many of these war wounds may also have been a big factor in lowering the incidence of war tetanus. The incidence of tetanus has steadily decreased with each war as medical care and prophylaxis have improved.

Comparable figures in civil life are not available. Certainly there are many people who sustain the same type of injuries found in this series who do not receive antitoxin and do not develop tetanus. Since antitoxin

Table V

Type Wound	No. of Cases	Deaths	Per Cent Mortality
Puncture	28	11	39.2
Incised	9	4	44.4
Lacerated	12	5	41.6
Contused	4	3	75
Gunshot	1	1	100
Septic	5	3	60
Burns	1	1	100

has not materially decreased the mortality of tetanus, a critical study of the value of prophylactic antitoxin is indicated.

Considerable work on tetanus toxoid has been done during the past few years and reports indicate that it may supplant the tetanus antitoxin derived from horses and cows, thus avoiding serum reactions. The

Table VI

Units A.T.S. Received First 48 Hours after Admission	No. of Cases	Deaths	Per Cent Mortality
None	3	2	66.6
(x)-Phenol intra. "v"	1(x)		
None to 10,000	13	6	46.1
10,000 to 25,000	10	6	60
25,000 to 50,000	18	7	38.8
50,000 to 100,000	16	7	43.7
Total	60	28	46.6

alum precipitated toxoid seems to confer an active immunity after several weeks and will probably be combined with antitoxin in the future. It is being administered with typhoid vaccine in certain divisions of the French and English armies. Its action is too slow for the treatment of acute tetanus, but would seem indicated as a prophylactic measure in compound fractures, gunshot wounds, or before some old traumatic wound is reopened.

The importance of incising the site of infection and removing all foreign material when present has been urged by many writers. At the Charity Hospital in New Orleans all tetanus cases are sent to the operating room before admission to the wards. Gage and DeBakey report a mortality of 20 per cent in a series of fifteen patients so treated.[4]

TABLE VII

	No. of Cases	Deaths	Per Cent Mortality
Wounds treated surgically	7	1	14.2
Wounds not treated surgically	53	27	50.9
Total	60	28	46.6

In this series some surgical procedure was instituted in seven cases with one death (mortality 14.2 per cent). There were fifty-three patients whose wounds did not receive any surgical procedure, with twenty-seven deaths (mortality 50.9 per cent). (Table VII.) Three of the seven patients who had incision and drainage of the initial wounds had a foreign body in the wound, three had a localized abscess requiring drainage, and the seventh patient had a suppurative arthritis of the knee joint following an incised wound (ax) one month previously. Open drainage of the joint with resection of the articular surfaces was done and thirteen days later tetanus developed along with high fever. The patient died five days later of tetanus. Autopsy showed some necrosis of the bony surfaces of femur and tibia.

One patient who had several foreign bodies in a wound developed tetanus following a fall on a gravel path. The abrasions apparently healed and the patient received a total of 250,000 units and avertin and left the hospital a month after the injury. After being up for several days, abdominal rigidity, trismus and convulsions recurred. There was some swelling about the initial abrasion and a small abscess cavity containing two pieces of gravel was incised and drained and the rigidity cleared up in several days without further treatment. This case illustrates the transient and even questionable value of antitoxin, since this boy had 250,000 units and yet several days later developed tetanus again when there was a flare-up in the original wound. Removal of the gravel when the patient was first seen probably would have decreased the amount of expensive antitoxin used as well as the time in the hospital, and have prevented the recurrence.

TABLE VIII

SURVIVAL PERIOD IN HOSPITAL

Time of Deaths	No. of Cases
1–24 hours	10
24–48 hours	5
3 days	6
4 days	3
5 days	1
6–16 days	3
Total	28

The importance of the wound care is again emphasized in the one patient of the sixty who would have been expected to die but recovered. This boy of 10 cut his leg on a piece of glass and the wound was sutured at once. Three days later (shortest incubation period in the group) trismus and convulsions developed. The wound was reopened, irrigated and wet dressings applied. The child had high fever, tachycardia, and all the unfavorable prognostic signs, but did recover.

Failure to drain a localized infection containing tetanus bacilli may result fatally in spite of long incubation and survival periods and over 200,000 units of antitoxin. A boy of 17 injured his hand with a blank cartridge and a week later developed mild tetanus and entered the hospital. He received large daily doses of antitoxin, but became progressively worse and died sixteen days after admission. Autopsy showed a palmar abscess containing powder granules, pus, and tetanus spores.

Twenty of the twenty-eight patients who died had wounds so located that incision or

excision could have been done. Half of these died within twenty-four hours after admission, however, so it is questionable whether any measure would have been of benefit. The other ten patients lived from two to sixteen days. (Table VIII.)

It would seem desirable to incise the infected wound and convert it into an open wound in which anaerobiosis would be discouraged if not prevented. If further toxin formation can be prevented after admission to the hospital and the free, unfixed toxin neutralized, some of the patients who live four or five days in the hospital may be saved.

Of the four possible modes of administration of antitoxin the intravenous and intramuscular routes are preferable. There is little evidence to show that intraspinal injection of antitoxin decreases mortality, and Wainwright states that "the best way to increase the mortality of tetanus is to give the antitoxin intraspinally."[4] In this series antitoxin was given intraspinally in so few instances that conclusions as to its value cannot be determined.

The amount of antitoxin given seems to make very little difference in mortality. Deaths were slightly less in the group receiving 25,000 to 50,000 in the first forty-eight hours. (Table VI.) Over half of the deaths occurred within twenty-four hours after admission to the hospital. All but two of these fifteen, however, received over 10,000 units on admission. Over 43 per cent of all patients who died received more than 50,000 units within a few hours after admission.

In this series avertin was used only in those patients having clonic convulsions; half recovered and half died. Both experimentally and clinically, convulsions seem to hasten the spread of toxin and their abolition would therefore seem important. Avertin is probably the best basal anesthetic available at present.

Phenol intravenously was given to seven of the sixty patients, with two deaths. The two who died had short incubation, while those recovering had incubation periods of over ten days. One patient with an incubation period of sixteen days received phenol alone and recovered. No one has ever repeated the excellent results obtained by Bacelli with the use of phenol.

Almost half of the wounds were puncture wounds, but incised wounds had a higher mortality. (Table V.) Many of these incised wounds had been sutured, or at least seen by a physician. There were three cases of tetanus neonatorum with two deaths. Two cases of tetanus followed attempted abortion. (Table III.) One was produced with a chicken feather and this patient died on the ninth day; the other patient recovered.

Certain signs and factors indicate a grave prognosis. A few are briefly listed: (1) an incubation period of less than a week; (2) wounds on the face, trunk, and upper extremities; (3) high fever and tachycardia; and (4) convulsions.

SUMMARY

Sixty cases of tetanus with a mortality of 46.6 per cent are presented. Prophylactic antitoxin did not prevent tetanus in 5 per cent of the cases. Patients receiving some surgical treatment of the infected wounds were four times as likely to recover as those not receiving any surgical care. The immediate administration of 50,000 units of antitoxin intravenously and the simultaneous administration of toxoid at five day intervals for two doses would maintain the blood antitoxin level at an adequate level.

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LOCAL ASPHYXIA AND TUMORS

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THIS research originated as one phase of a frustrated investigation of circulatory and vascular disturbances. The production of diabetes by the inflammation following temporary ligation of the pancreatic vessels was first reported,[1] and chronic hypertension was obtained with similar treatment of the kidneys[2] prior to the work of Goldblatt.[3] The tumor research would have been facilitated if other parts of the general investigation could have been finished.

About 1917, when the lymphocytic theory[4] of tumor defense was prominent, the writer mentioned to Dr. J. B. Murphy the intention of trying to excite a local tissue reaction around tumors. This field is still untouched. Bowen's[5] permanent ligation of the blood supply to the tumor region is entirely different, and Lumsden's[6] temporary ligations were made for testing the effects of a serum and were continued only for two hours, so that the full effects of asphyxia were missed. Sufficiently prolonged asphyxia of a typical viscus such as the kidney or testis produces complete atrophy and sclerosis, the parenchymal cells dying first, presumably because of their specialization or high metabolism, while the connective tissue of supposedly lower metabolism survives. In applying this method to tumors, the question arises whether the tumor cells will die quickly because of their high metabolic activity with the accompanying need for oxygen and accumulation of lactic acid[7] and other products, or whether their capacity for anaerobic metabolism[8] will endow them with a special resistance to asphyxia, or how they will be affected by the high oxygenation[9] of the blood during the subsequent reactive hyperemia or by the local metabolic conditions of inflammation.[10] Second, owing to the importance of the tumor stroma, there are questions of the influence of capillary hemorrhages, round cell and leucocyte invasions and fibrosis, all of which can be produced by asphyxia and which are commonly associated with tumor regression. Finally, the altered interchange between tumor cells and blood, the stimulation of local or general immunity reactions,[6,11] and many indefinite possibilities may be considered in relation to any possible theories of tumor growth.

PLAN AND METHODS

This work has involved the use of several thousand animals, chiefly rats, and has extended over ten years. Tumor-bearing animals for starting the various mammalian tumor strains mentioned were purchased from the Crocker Institute. Except as otherwise mentioned, the tumors were implanted in the lower thigh or upper calf. The use of rubber bands as tourniquets, and the reactions in normal animals, comprising hyperemia, paralysis, shock, the endurance of asphyxia by limbs and viscera, the influence of temperature, and numerous other details have been described in a series of previous papers.[12] The results are here presented in compressed summary.

I. Immediate Effects of Asphyxia. 1. *Reaction of Hemorrhagic Congestion.* With a tumor in the usual location under the skin or amid the muscles, ligation of a leg is followed by a striking circulatory reaction. The pink hyperemia of the normal parts changes to more or less of a bluish tinge over the tumor. After the longest ligations, when the color of the foot is a rather bright red, the tumor area shows a deeper blue, darkening gradually to almost black. Incision shows that the normal parts are edematous and also surcharged with bright arterial blood. On the contrary the tumor is swollen and darkly engorged. This in-

tense hemorrhagic congestion may involve the entire tumor tissue but is not confined strictly to it, for the maximum engorgement is often found in a narrow zone comprising the outermost layer of the tumor and a very thin layer of surrounding tissue. This reaction, in its striking contrast of blue against red, may be somewhat less readily induced in certain locations such as the foot or groin, but in general it seems to be a constant characteristic of all the tumors studied, in comparison with the normal peripheral tissues of the body.

2. *Necrosis of Tumor Tissue.* Typically, the dark blue color acquired by the tumor soon after release of the ligature changes gradually in the course of a few hours to black, and if the ligation has been present long enough the tumor softens and sloughs out partially or completely. This process naturally occurs most readily in the center of the tumor mass, where the nutrition is poorest. The better nourished peripheral layers, though they may have been most deeply congested, survive much more stubbornly and often continue to grow around the margins of the ulcer. Not only partial but also complete destruction is produced somewhat more easily in large tumors than in small ones, for one of two hypothetical reasons: (a) the enzymes and toxic products from the dead central part may poison the outer layers; (b) the small tumors may be in closer relation with a normal blood supply, while larger tumors may be more dependent upon their own vascular apparatus, which is insufficient and breaks down under congestion. The normal tissues meanwhile display merely edema and inflammation, and unless the ligation is carried to the extreme of producing general gangrene, there is always a sharp demarcation between the necrotic tumor and the inflamed surrounding tissue.

3. *Metastases.* From the outset, the possibility was borne in mind that a procedure which produces such marked hyperemia and edema may conceivably augment the metastatic transport of tumor cells, either through the blood stream or through the lymph. The impression gained from hundreds of observations is that such an occurrence is absent or slight. Tumors subjected to various degrees of partial damage by ligation have apparently not metastasized sooner or more extensively than untreated tumors. Complete destruction of a tumor has been followed by metastases only when there was reason to suspect a transportation of tumor cells prior to the ligation. Doubts of the absolute validity of these statements have arisen only lately in regard to the Walker tumor, and can perhaps be explained on the assumption that the growth of existing metastases may be accelerated when the original tumor is nearly or completely destroyed.

II. Results in Mice. Experiments were undertaken with three tumors from the Crocker Laboratory, namely the sarcoma 180, a carcinoma and a chondroma. Owing to unfavorable conditions the mortality among mice was so high that this phase of the work was curtailed. All tumors were allowed to grow to 0.75 to 1.0 cm. diameter before treatment.

The reaction of hemorrhagic congestion was very marked, and ligations of three to four hours, or sometimes as short as two hours, resulted in complete necrosis and healing without recurrence, in the case of the sarcoma and carcinoma. It would have been desirable to repeat these results in more numerous mice of different strains and at different seasons, in order to establish more positive conclusions.

The chondroma is known as a tumor with nearly 100 per cent of "takes," a very slow growth rate, and benign character, which finally proves fatal through mere increase in size. Hemorrhagic congestion following ligation seemed to be distinctly less than in the preceding tumors. According to Neuberg and Caspari,[13] chemotherapy also causes less congestion, hemorrhage and necrosis in benign than in malignant tumors. This tumor survived longer asphyxia than the preceding ones, and the exact time requirement was not established. The surviving tumors could be killed by

successive repetitions of ligation, so that no actual failures were encountered when the animals lived long enough.

It is open to speculation whether the differences between the malignant and benign mouse tumors, also between mouse and rat tumors, are connected with rapidity of growth, metabolic activity, vascular fragility or other characters.

III. Single Ligations in Rats. The tumors used were the Flexnor-Jobling carcinoma, the Crocker sarcoma 39, and the Walker tumor. Treatment was given when they had reached a diameter of 1.5 cm. or considerably larger. The dangers from shock, ulceration, infection and cachexia increase with the size of the tumor. All these troubles would be less with tumors of similar size in a larger species, but in the rat the mortality from these accidental causes multiplies the labor of obtaining a sufficient series of survivals for a convincing length of time.

Large tumors were used in the face of these difficulties for two reasons: (a) because the attempt to cure a well advanced malignant growth is believed to be the best test of any treatment, and (b) as assurance against spontaneous recessions.[14] Tumor specialists estimate standard percentages of recessions for different tumor types, in a traditional location along the side of the body. It is likewise evident that these tumors grow in a nodular mass almost separate from the body, where they can receive nutrition only from tiny subcutaneous vessels and a thin layer of superficial muscles. Recessions are thus so readily accounted for by mechaniccal deficiencies of circulation that it is only surprising that they are not more numerous. Tumors in the mesentery or omentum generally grow from only a narrow pedicle and thus are still more subject to necrosis, often having only a thin shell of living tissue which is perhaps nourished by lymph diffusion and which is likely to be found non-transplantable. On the contrary, a tumor well established in the liver, kidney or spleen of a rat, with an abundant blood supply, has less necrosis and is never cured by any spontaneous process. Leg tumors have available nutrition from larger muscles and blood vessels, and they are compressed by the tighter skin so that they grow from a broader and firmer base than axillary tumors. The present experience is believed to be large enough to support the declaration that tumors of the kinds here described are practically free from spontaneous recession, and that the figures given for cures are absolute and not subject to statistical revision.

TABLE 1
RESULTS OF LIGATION OF RAT TUMORS

Tumor	No. of Rats	Hours of Ligation	Results	
			Recurrence	Permanent Cure
Sarcoma	42	5	16*	26
	31	6	7	24
	23	7	0	23
F-J carcinoma	37	5	27	10
	33	6	15	18
	19	7	0	19
Walker tumor	18	5	18	0
	16	6	15	1
	14	7	8	6
	9	9	0	9

* In 3 of these cases, a second five-hour ligation brought complete cure.

With the tumor in the lower part of the thigh, a tourniquet about the upper thigh can be tolerated for several hours without fatal shock. For longer ligations it is necessary that the tumor be in the foreleg or foot, so that the tourniquet can be placed lower. Previous papers have shown that it must be placed below the knee in order to avoid fatal shock for periods long enough to test the maximum endurance of asphyxia by normal tissues. The normal tissues can thus survive approximately 13 hours of deprivation of circulation, and there are plain signs that they could survive several hours longer except for the secondary consequences of ulceration and thrombosis at the ligature site, which are more

serious in rats than in larger species. A tumor-bearing leg cannot survive more than eleven or rarely twelve hours of ligation, because the ulcerating tumor opens a channel for infection which the damaged normal tissues cannot resist. Also the longer the ligation the greater is the tendency of rats to gnaw the anesthetic limbs. It was sometimes necessary to prevent this, and also the trauma of dragging the legs around, by confining them for several days in individual boxes too narrow to permit turning.

Repetitions of the experiments in an attempt to increase the proportion of cures gave a considerably poorer result than that shown in the table. Many such series were carried out with irregular results, which seemed to vary with the season, with different strains of rats, or other unknown variables. Lengthening the time of ligation beyond seven or eight hours yielded no benefits to compensate for the increased danger. This result is in harmony with the view that the curative process consists not in direct asphyxial injury of the tumor cells but in a special vascular or tissue reaction.

No plan of ligation was ever found which cured more than a variable and sometimes small minority of rat tumors. The differences in resistance of the three tumor types were uniform, in that the sarcoma 39 was least resistant, the Walker tumor most resistant, and the F-J carcinoma intermediate. The Walker tumor may have undergone changes;[15] it was totally incurable by ligation in the later stages of the work.

The manner of tumor death by dark hemorrhagic congestion and abrupt sloughing is entirely different from spontaneous recession. The center of any large tumor breaks down with the briefest interruption of the blood supply. The peripheral layers are more resistant in proportion as they are better nourished. After long ligations, when no tumor tissue can be found in the ulcer by gross inspection, nests of cells are sometimes found microscopically adjacent to normal tissues. Serial sections were never made, but after some of the longest ligations ordinary slides failed to reveal any living tumor. The smallness of the residue was further demonstrated by the fact that the ulcer sometimes healed completely or almost completely before a recurrence appeared. The inflammatory picture following ligation was such that one pathologist accustomed to tumor studies assumed at first glance that it was the result of a heavy *x*-ray dose. The results were apparently not due to accumulation of any special type of defensive cells, as the tumor death occurred suddenly before any such accumulation was possible. One possible interpretation is that the tumor stroma easily breaks down, and that the most resistant tumor cells are those which are still supplied from normal blood vessels by reason of their contiguity to normal tissues.

IV. Repeated Ligations. The production of diabetes by progressive injury of the islands of Langerhans, and of chronic hypertension by nephrosclerosis, are examples of the degenerative and sclerosing effects of repeated brief clamping of the blood vessels of viscera. It was hoped that some such effects might be produced by repeated ligations of tumors; in particular, that a gradual sclerosis or some closer imitation of spontaneous recession might thus be obtained instead of the sudden necrosis which follows a single long ligation. As any ligation plainly damages a tumor out of all proportion to the normal tissues, there was reason to hope that repetitions of the process would increase this selective effect and also reduce the dangers. Also edema, hyperemia, cellular and tissue reactions, etc., may be kept active over a longer time by repeated than by single ligations.

Preliminary experiments comprised ligations of legs of normal rats for longer or shorter periods at intervals of hours or days during several weeks. The main outcome was that the peripheral tissues proved to be much more resistant to this process than the viscera; the desired tumor treatment is thus feasible without serious injury of the normal parts. A former plan of trying to produce chronic local edema or sclerosis in larger species could not be carried out. In

rats such an attempt encounters the difficulty that the skin is the point of lowest resistance, and the experiments when pushed too far are stopped by ulcerations. Minor degrees of permanent atrophy of the limb were sometimes produced. Naturally, short ligations can be repeated at the shortest intervals, while long ligations require correspondingly lengthened rest periods.

Rats bearing the three types of tumors, which escaped accidental death and survived long enough for complete judgment of the results, were divided into four groups as follows:

1. Short ligations (fifteen to forty-five minutes) with relatively long intervening periods (one day to one week); twenty animals. These attempts to excite slight recurrent hemorrhages, cellular reactions or fibrosis in tumors were soon abandoned, because the tumors merely ulcerated while continuing to grow rapidly.

2. Longer ligations (one to four hours) at long intervals (one to two weeks); forty-four animals.

3. Long ligations at shorter intervals (one to three days); fifty-nine animals.

4. Eclectic ligation periods, beginning with short and increasing to longer times, or vice versa, and repeated according to the apparent limit of the individual rat's strength.

The tumors thus treated all ulcerated but continued to grow. No fibrosis and nothing resembling spontaneous recession was ever produced. As these rat tumors are practically never (only three instances in this entire research) killed by a single ligation of less than five hours, so also ligations of four or four and one-half hours, repeated one to four times or as often as the rat's strength permitted, were followed only by more or less complete exulceration and later recurrence. The failure to obtain a single cure in these series adds to the evidence that tumors of this size are dependable test objects and that cures when obtained are genuine.

Near the close of the research, attention was given to the possibility that the failures in these attempts might be explainable by unduly long intervals between the ligations, allowing time for the active inflammation to subside. A fifth series was then undertaken, with ligations of one-half to one and one-half hours' duration, repeated at intervals of one to two hours throughout two days. In this way six cures were obtained among fifteen tumors. Furthermore, certain of these tumors were as large as 3 cm. in diameter—larger than can be treated successfully with single ligations because of the danger of shock. The successes were all among the sarcomas and F-J carcinomas. All the Walker tumors finally recurred. The inflammation of the normal tissues and hemorrhagic congestion and necrosis in the tumor were the same as with single long ligations. It was impossible to make up a larger series because of the ending of the research. The method seemed to promise better success and greater safety than single long ligations. Its theoretical importance is as evidence that prolonged continuance of asphyxia is not a necessary condition for tumor destruction.

SUMMARY AND CONCLUSIONS

This paper describes the effects of local asphyxia upon transplanted tumors of rats and mice located in peripheral regions such as the limbs.

1. The first effect is the so-called reaction of hemorrhagic congestion, in which the tumor turns dark blue in contrast to the bright arterial hyperemia of the surrounding normal tissues.

2. Subsequently, the tumor undergoes extensive or complete necrosis, actual cures by this process being more frequent in mice than in rats.

3. These results of prolonged single ligations can be duplicated or even improved by suitable repetitions of brief ligations. The most important theoretical deduction is that the tumor death is not due to the simple duration of asphyxia or toxic action of accumulated asphyxial products.

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SPASMODIC TORTICOLLIS: ITS CAUSE AND TREATMENT*

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A STUDY of the literature of so-called "spasmodic torticollis" reveals confusion and uncertainty as to its etiology as well as hopelessness in the prognosis and treatment. Neurologists and neurologic surgeons have failed to agree as to the character and location of the processes in the brain, responsible for the external evidences of the dystonic disturbance. However, other facts arrest the attention, especially the clinical descriptions of the affection and the reports of the pathologic findings in the few cases that have been studied post-mortem.

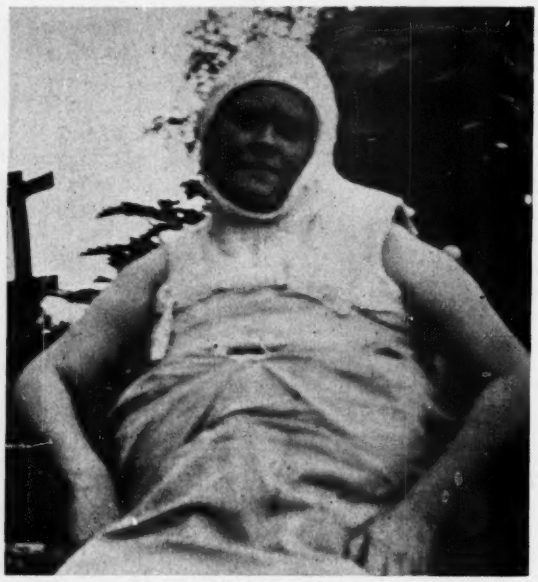

FIG. 1. Cast as applied.

An outstanding and most disturbing symptom has given the name to the condition and partly accounts for much of the confusion existing in its discussion and literature and has hindered accurate description and uniform understanding of this most distressing ailment. Even before any pathologic changes had been demonstrated by post-mortem studies, two theories as to the etiology were extant, namely, the psychogenic or as it is sometimes termed, the functional; and the organic, in which structural changes were predicated in the central nerve system. These classifications still obtain despite the disparity between the clinical evidences of two patently differing conditions.

The first, or psychogenic, theory postulates abnormal emotional reactions in a patient or subject of distinctly neurotic type in whom, as a consequence of some mental or physical (or both) stress or fatigue, muscle spasms occur. The inherent instability of the system (either congenital or acquired) renders the nerve cells in the base of the brain (which supply the eleventh cranial and the first and second cervical nerves) more susceptible to the external stimuli of occupation or surroundings, and result in their hyperactivity. In other words, it is a basically functional weakness of the nerve cells, which, under severe mental or physical stress gradually produces a chronic spasm of the muscles motivated by them. In the development of this theory, it is highly probable that the cells are in a state of debility or exhaustion from overuse which renders them more susceptible to irritative and stimulating external conditions, but which is not followed by *degenerative* or *pathologic changes in the cells.*

The second, or organic, theory predicates a pathologic lesion in the basal ganglia of the brain, including the striatum, the capsule, the basal nuclei or "a disturbed labyrinthine function due to a unilateral lesion of the brain stem" (Grinker[1]). Grinker also believes that the condition involves the capsule and at times the adjacent nuclei. Post-mortem examination in several cases has shown pathologic

* Presented at the meeting of the American Orthopedic Association, June 6–8, 1939, Buffalo, New York.

changes in the "large cells of the caudate and putamen" (Alpers and Drayer[2]).

The writer believes it entirely conceivable that the long-standing and persistent cases of the psychogenic type of pure and uncomplicated spasm *might* develop secondary degenerative changes in the central motivating cells but thus far there have been no published results of post-mortem findings in such cases and no clinical evidences of central pathologic complications in this type. In two of the cases treated, the spasm had been present for eight or more years yet there was no evidence of muscle or fibrous tissue changes and complete relief was obtained by the method of treatment to be described. The position of the head in all of these cases is analogous to that of the ischemic or traumatic wry neck commonly seen in childhood and following injury to the sternocleidomastoid muscle, but differs in that (1) the spastic type presents no structural shortening of the muscles or infiltration of the intramuscular fasciae such as is always present in the traumatic type, and (2) the contraction is clonic and not constant.

From the series of cases treated, the writer believes there are two distinct types of the affection and that both theories are correct, each in its respective type. Several of our earlier patients were completely relieved by prolonged fixation, without any relation to underlying pathology but purely with the idea of giving relief from the pain and spasm. However, the results of the treatment and more intensive study of the symptoms of the cases evidenced a marked clinical difference between the two types and also suggested the probable cause of the failures following surgical and other methods of treatment. The differentiation of the two types is made from the character of the symptoms present. In the pure or psychogenic type of case, there are no other symptoms than the clonic spasm of muscles, singly or in groups, while the organic type presents symptoms referable to degenerative changes in the motor cells. These changes are characterized by athetoid movements in various parts of the head and neck. Thus, it was felt that, in the psychogenic cases, there was probably some local functional disturbance rather than a degenerative condition in the motivating cells. This distinction has been the criterion in the selection and treatment of our cases. In several of our patients, the spasm had persisted for six or more years, but no other symptoms have developed. In the organic type, however, the athetotic movements developed early with the torticollis spasm and gradually involved various parts of the upper body such as the tongue, neck, arms and hands and in these cases

Fig. 2. First celluloid brace.

Fig. 3. Second brace.

Fig. 4. End result.

it was felt that failure would follow the measures which proved successful in the others. We believe there a sharp distinction must be drawn between the two types and that the so-called psychogenic should be known as the true spasmodic torticollis while those cases which present other symptoms characteristic of degenerative changes in the basal ganglia and cells or in other parts of the base of the brain, should be classified by a title descriptive of the pathology in those parts.

Fig. 5. Another type of brace to follow cast.

Another strong argument in favor of this distinction lies in the results of the treatment. Heretofore, the common name of spasmodic torticollis carried with it a hopeless outlook for relief. Most writers claim that all methods of treatment, whether mechanical, electrical, surgical or physiotherapeutic, were failures or left the patient almost as badly crippled as before (this is especially evident after neurectomies). It is highly probable that where failure resulted, it was due to a lack of differentiation between these two outstanding types of cases. Stimulated by the failures of surgical and other measures and impressed by the theory of psychogenesis as exemplified in other conditions, we were led to feel that we were dealing with a badly balanced nerve system, which under the stress of life, occupation or environment, developed an asthenia or exhaustion of the basic cells which in turn rendered them hypersensitive to external stimuli thus reflexly setting up spasms in the muscles motivated by them.

Fig. 6. End result in 46 year old patient with most resistant type.

In 1863, Hilton (London) published his lectures on "Rest and Pain," a book which remains a classic to the present day and provides a basis for the successful treatment of many more recently recognized ailments. He demonstrated that the mechanical and physiologic rest of parts, which are or have been subjected to injury or strain, was one of the most potent factors in the restoration of health to those parts. Acting upon the principle that the central cells and the muscles motivated by them complement each other, we decided to try placing the central cells at rest through immobilization of the parts which they supply. This principle of treatment is not new. It had been tried in this condition many years ago but was discarded as unsuccessful. We believe that the reason for failure in these attempts was either a lack of persistence on the part of the physician and patient in carrying out the plan of treatment or failure to recognize the dif-

ference in the two types of torticolli. More rapid methods of relief or cure have also been tried, but they too have failed utterly or have left the patient with a loss of head and neck control fully as dire as the original malady.

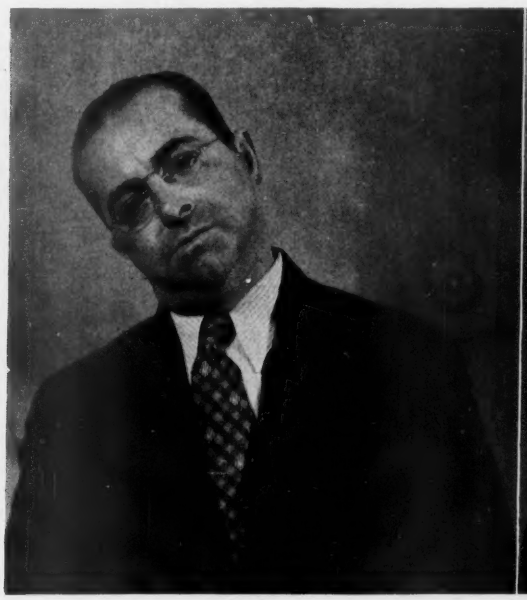

Fig. 7. Before treatment.

Fig. 8. Two and one-half years after treatment had been started.

Profiting by the markedly beneficial results of the complete physiological rest to the nerve cells in cases of completely or partially paralyzed muscles in infantile paralysis, through the removal of all stimuli from those cells by external fixation of the motivated parts, we felt that if the head and neck muscles could be securely and firmly fixed by plaster of Paris for a sufficient length of time, the basal cells supplying them would be placed at rest. With an opportunity to recover their tone, the cells would lose their hypertonicity.

The coöperation of patients over varying periods of time has allowed us to use the method. Results have proved the correctness of the theory. In one patient, a woman 47 years of age, the spasm of the muscles was so great that an anesthetic was necessary for holding the head straight while the cast was being applied. This patient developed a pressure sore on the cheek within three days, making it necessary to cut out part of the cast. However, she persisted with the treatment and after two and one-half years of fixation, was relieved of all spasm and remains well today (after twelve years).

For the application of the cast, the patient is preferably seated on a stool, the hair is cut short and stockinette is slipped over the head and well down on the chest, holes being cut for the arms. Cotton wadding is then placed about the body, neck and head. An assistant holds the head in as correct position as possible, the arms are held extended laterally at a right angle and plaster bandages are wound snugly about the body, neck and head. Reinforcing slabs are placed from the top of the head down the back and pressed closely to the neck. The bandage covers the entire head, but leaves the face open. When the plaster has set firmly, the cast is trimmed about the face and about the lower edge of the jaw, allowing a slight space for jaw action. The ears are then uncovered and the armholes carefully trimmed. This cast is worn for at least six months and sometimes a year. In hot weather, it should be changed every six months, great care being used to preserve the corrected posture. The comfort following the arrest

of the spasms insures coöperation of the patient in spite of the discomfort of the weight of the cast and the odors from the skin. After about two years, when the third or fourth cast is removed, the patient is able to hold the head quite still and there is seldom any pull of muscle except in the presence of excitement. A celluloid cast is then made over a plaster mould and so cut that it can be removed for bathing. This must be worn continuously for four or five months and can then be gradually removed for the application of a light steel support which provides a sense of fixation rather than its reality. Within a few months more, this is gradually laid aside. In excitement or unusual fatigue, these patients may have a slight sense of tension, but this quickly disappears and in none of our ten cases has there been a recurrence.

Fig. 9. Celluloid and metal splint following plaster of Paris.

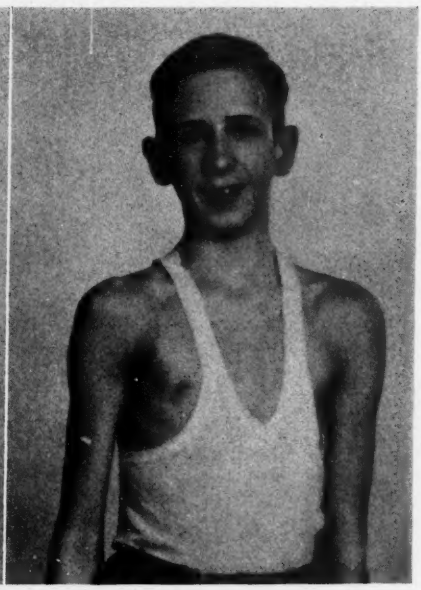

Fig. 10. End result after three years of treatment.

In one case, a boy of 13, suffering from mild congenital spastic diplegia involving the entire body, there was distinct torticollis and associated athetosis of the head, neck and arm muscles. His activities were greatly impaired and the uncertainty of his movements prevented him from joining in the sports of his playmates. Inasmuch as there still remained three or four years for growth and development and as the influences of these processes often exert a most beneficial effect upon the spastic condition, it was decided to try the fixation treatment. Plaster of Paris was used (about the head and shoulders) for two years and this was followed by a removable celluloid and metal brace. The results have been beyond our expectations. The patient has gained greatly increased control of his head and arms. Now when the brace is removed, he is able to control these parts much better and they show much less spasticity than previously. In addition, the boy's mental reaction to his surroundings has been greatly improved and he is rapidly losing the inferiority complex that formerly handicapped him. He plays ball and has even joined in football with his schoolmates. Although there is evidence of steady improvement, it is not to be expected that the congenital brain changes will be completely overcome. The prolonged rest has, however, been of great value and there is every reason to feel that further growth will promote greater improvement in his condition.

SUMMARY AND CONCLUSION

This presentation offers three main points regarding spasmodic torticollis: (1) the careful differentiation between two patently separate conditions which present one outstanding symptom in common; (2) the need for efficient and prolonged fixation, preferably by plaster of Paris, until spasm is overcome; (3) the necessity of absolute coöperation on the part of the patient in following out the treatment.

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The reflex of the tensor fasciae latae is unaffected in sciatic neuritis, but is usually lost in affections of the lumbosacral roots.
From—"The Injured Back and Its Treatment" by Ellis (Charles C. Thomas).

SURGERY IN SYPHILITIC WOMEN*

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SYPHILIS in its relation to surgery brings up problems which need investigation. Textbooks on surgery mention these problems meagerly, and the literature fails to give comprehensive material for clarification. The problems with which the surgeon is confronted are as follows:

1. Differential diagnosis.
2. Prognosis:
   (*a*) Surgical risk.
   (*b*) Prevention of surgical complications.
   (*c*) Prevention of infection of the part of the surgeon.
   (*d*) Comparison of recovery and wound healing between syphilitic and non-syphilitic patients.

It frequently happens that through errors in diagnosis, patients with syphilitic manifestations are subjected to unnecessary operation. The gastric crisis of tabes dorsalis may mimic a surgical abdomen. Muzum collected a long list of needless operations of this kind. Fabian reported a case in which what seemed to be metastases, appeared below the left costal arch five years after an ovarian tumor had been removed. A Wassermann taken was positive, and, after anti-syphilitic treatment, the growths disappeared. These tumors and the original ovarian tumor were gummata. Gellhorn cites a case where at operation he thought he was dealing with an inoperable carcinoma of the liver; anti-syphilitic treatments cured the patient. In many instances chancres or gummas of the cervix have been mistaken for carcinoma.

Uterine hemorrhages may be caused by syphilitic changes in the uterus.

Plastic peritonitis may be due to chronic syphilis, and many operations for adhesions, chronic appendicitis, and diseased pelvic organs are performed without a final cure resulting.

Stein and Hensel reported a case in which operation was done with the preoperative diagnosis of a large intra-abdominal tumor, ovarian cyst or pedunculated fibromyoma, or mesenteric cyst. Before operation the blood Wassermann was negative. At laparotomy the abdominal cavity was empty. The tumor was a suppurating large gumma of the abdominal wall. The spinal fluid Wassermann was positive after the operation; abnormal reflexes pointed to tabes dorsalis. With thorough anti-syphilitic treatment the tumor disappeared in about two months.

Recasens writes of a case of syphilitic endometritis, a rare occurrence, in which he had operated upon by error, performing a vaginal hysterectomy.

All these examples show how important it is to make a differential diagnosis before operation. At the House of Detention for Women of the City of New York, a Wassermann and a check test are performed routinely. Because these procedures are not always followed in private institutions, such mistakes as listed above may occur. Wassermann or other tests, spinal fluid examination, dark field and histologic examinations will decrease surgical errors. A trial of anti-syphilitic treatment in suspicious cases is a wise procedure even when Wassermann and all other tests are negative. Keen judgment helps to prevent surgical errors.

As to the question of *prognosis and surgical risk*, differentiation must be made between unrecognized, untreated cases of syphilis, and treated cases. In the first group the prognosis must be considered theoretically poor. It is recognized that spi-

* From the House of Detention for Women of the City of New York, Department of Correction Hospitals; Director of Surgery, Edward C. Brenner, M.D., F.A.C.S.

rochetes spread through blood and lymph vessels very rapidly and cause a general systemic infection. Almost every part of the body is affected, especially the circulatory system, heart and aorta. The kidneys and brain frequently remain damaged even after treatment. A syphilitic cachexia may develop—especially in young women. Insurance companies rate individuals who have had syphilis in the second class.

Many postoperative complications due to syphilis are described in the literature. In one, after a fibroid uterus had been removed, the wound did not heal and the diagnosis was not made until a gumma developed. Specific ulcerations and extensive suppurations may develop in a wound or incisions may open up without suppuration. All these wound complications may readily react to specific treatment. However, operative mortality from cerebral hemorrhage of syphilitic origin may rise. General anesthesia might conceivably affect brain pathology, while spinal anesthesia is rejected by some surgeons because of the possibility of exciting an old syphilitic meningitis.

Prognosis and surgical risk are much better in old treated cases of syphilis than in untreated cases, as our statistics show. At least two weeks preoperative treatment should be instituted. In acute surgical cases, if there is no time for such treatment, it should be given during convalescence. Patients with old syphilitic organic lesions, with severe anemia or cachexia, etc., are poor risks, and operation should, if possible, be deferred.

Brief mention should also be made of the possibilities of infection during operation. The physician, his assistant, or an infected instrument may occasionally be the cause of inoculating a patient with lues. The surgeon can also inoculate himself after some injury, perhaps so slight that nothing is noticed until secondary lesions appear. Untreated patients may infect the surgeon, but it is probable that even small amounts of anti-luetic treatment will render the disease non-infectious.

The material here presented is drawn from a penal institution. From 1933 to 1938 inclusive, there were 775 operations in the House of Detention for Women of the City of New York. The greater number were minor operations which are eliminated. We have selected for comparative study the more important major operations, numbering 164. The ages of the patients ranged between 20 and 62, but most were in their thirties.

All patients had a venereal check-up in which at least two types of laboratory tests for syphilis were performed. All syphilitic patients received preoperative treatment consisting of at least six neosalvarsan and ten bismuth injections. Women with untreated syphilis were not operated upon, except in emergencies. Anti-syphilitic treatment was resumed in all cases during convalescence.

In these 164 cases there were eighty-three instances of old syphilis treated preoperatively, and eighty-one with no evidence of the disease.

The diagnoses among these 164 patients were:

Adnexal conditions	56
Fibromyoma of uterus	48*
Large cystorectocele and lacerated cervix	13
Ovarian cysts	7
Retroversion	3
Rectovaginal fistula	1
Tubal pregnancy	1
Prolapse of uterus	1
Torsion of uterus causing acute surgical abdomen	1
Gall-bladder conditions	9
Thyroid conditions	6
Hernia	3
Carcinoma of breast	1
Acute appendicitis	4
Acute intestinal obstruction	2
Kidney conditions	2
Eye conditions	4
Ear condition	1
Orthopedic condition	1

* About 50 per cent were complicated by adnexal or other conditions.

The types of operation performed were:

Hysterectomy	
Supracervical	43
Total	2
Conservative pelvic operation	71

Vaginal and cervical repair	13
Cholecystectomy	9
Cholecystostomy	1
Thyroid operation (usually subtotal thyroidectomy)	6
Exploratory laparotomy	2
Hernia repair	4
Appendectomy	4
Radical mastectomy	1
Enucleation of eye	4
Nephrectomy	1
Nephrostomy	1
Major orthopedic operation	1
Mastoidectomy	1

The operations were carried out under general anesthesia in most cases, but spinal was used in a few selected instances.

To evaluate the results of operation, we considered the mortality, the morbidity, and the wound healing as criteria, and we compared the infected with the non-infected groups.

We had three deaths in this series of 164 operations (1.82 per cent). Two had syphilis, one did not.

The morbidity may be a better criterion for comparison. Twenty-one patients had a disturbed postoperative convalescence, a percentage of 12.80 per cent. Of these there were thirteen old syphilitics and eight non-syphilitics. There was a slightly greater percentage of postoperative complications in infected than in non-infected women.

If wound healing is taken as a criterion of the operative results, the following data are pertinent:

	No.	Per Cent
Primary union	140	85.37
Old syphilitics	65	
Non-syphilitics	75	
Wound infection	24	14.63
Old syphilitics	16	
Non-syphilitics	8	

Most of the wound infections were slight. We also included among these wound infections, cases with original drainage during operation, healed by secondary union. Wound healing was apparently better in non-syphilitic cases, but in our series we could not attribute any wound infection to gumma or syphilitic ulceration.

It should be noted that among the operated cases, there were more than 50 per cent with a history of gonorrhea and about 15 per cent were drug addicts. These conditions might well influence the operative prognosis.

Table 1

Wound Healing	Total		Syphilitics 83		Non-syphilitics 81	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
Primary union	140	85.37	65	78.43	75	82.58
Wound infection	24	14.63	16	19.27	8	9.87
Postoperative morbidity	21	12.80	13	15.66	8	9.87
Mortality	3	1.82	2	2.40	1	1.23

The three deaths which occurred are briefly reported:

I. M. A., age 36, white, was admitted to the surgical ward with an acute exacerbation of a pelvic inflammatory disease. She had a high temperature and a leucocytosis of 18,000. She was in the hospital under conservative treatment for three weeks. Blood Wassermann was negative. During this time, the abscess ruptured and drained through the rectum. Not much improvement occurred and the patient was desirous of operative relief. Bilateral salpingo-oöpherectomy was performed. Postoperatively, the patient's condition was poor, and in spite of supportive treatment, she died in sixteen hours, possibly from shock. Postmortem examination was refused.

II. O. T., age 38, colored, was admitted to the surgical ward with a diagnosis of possible intestinal obstruction. The patient had an old syphilis, untreated in the institution. She was operated on October 14, 1936. Exploratory laparotomy and ileostomy were performed. She died the next day.

At autopsy there was dark reddish fluid, especially in the pelvis and in both flanks. The intestines were distended, partly reddish brown in color. Circumscribed peritonitis was present above pelvis.

Both lungs were adherent to the posterior pleural wall. The descending aorta showed beginning sclerosis in the form of yellowish patches. Adhesions were noted between the diaphragm and right lobe of the liver.

The loops of the ileum were situated in the pelvis and a small hole 1 inch in diameter was found in the mesosigmoid. Into this hole protruded several twisted loops of ileum. The margins were firm and showed no signs of fresh laceration. The mesenteric vessels did not show thrombosis.

The cause of death had been intestinal obstruction from abdominal hernia through a congenital defect in the mesosigmoid.

The above two deaths were surgical deaths. Apparently syphilis played no part in the final outcome.

III. E. S., age 45, white, was admitted to the hospital with the diagnosis of ovarian cyst, chronic cholecystitis, and cholelithiasis, vascular syphilis, chronic drug addiction, and chronic gonorrhea.

Her syphilis dated back to 1927. She had had four courses of anti-syphilitic treatment in 1936 and before operation she had a few salvarsan and a few bismuth injections. She had been a drug addict for fifteen years.

On June 16, 1938, left oöphorectomy and cholecystectomy were done under general anesthesia.

During convalescence, the following complications developed: bronchopneumonia; multiple abscesses in the left lung; empyema of chest. She was operated on again on September 3, 1938, when a left pneumonostomy was performed. Blood transfusion was given after this operation. The patient died on September 14, 1938 with signs of a pulmonary embolus.

Autopsy. In the right upper lobe, there was an abscess 1½ inches in diameter, with congestion and edema in this lung. In the left chest there was 600 c.c. of pus, and many abscesses in the left lung. The aortic walls were thickened at the bases of the cusps. The coronary arteries were slightly sclerosed. The descending aorta showed many linear "tree barked" areas, pearly gray in color and there was slight thickening of the aorta itself with scattered, raised, yellowish, atheromatous placques. The liver was congested with parenchymatous degeneration. The spleen was larger than normal. The kidney surfaces showed two large scars. The cortex and medulla were diminished, and several cysts were found in the parenchyma. The brain showed marked atrophy of the cerebral gyrae, and the gray matter was atrophic.

In this patient there was evident marked pathology of the organs as a result of the old syphilis. She had to be considered as a bad operative risk and probably the complications could be attributed to these pathologic changes. She had also been weakened by her drug addiction.

DISCUSSION

One hundred and sixty four major operations were studied. In each case at least two laboratory tests for syphilis had been done. With the exception of emergencies, every syphilitic patient was treated routinely before operation. There were no cases of acute syphilis in this series. The operative results were studied in two groups and compared on the basis of mortality, morbidity, and wound healing.

It is the consensus of opinion of the medical profession that patients with acute syphilis should not be operated, except in grave emergencies. This precaution is for the safety of both the patient and the surgeon.

By a routine Wassermann test many questionable diagnoses may be clarified and postoperative results are safeguarded. In addition, the surgeon will be protected against infecting himself on an unrecognized syphilitic patient. Cases of chronic lues should be given routine treatment.

CONCLUSIONS

Serologic tests for syphilis and the institution of anti-luetic treatment when indicated are advisable in all operative cases.

It would appear from this study that chronic syphilis as a complication to surgery presents only a slight risk as regards mortality, morbidity, and wound infection.

It is felt that major surgery in syphilitic patients can be done with comparative safety if the above mentioned precautions are followed.

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In cardiovascular-renal disease in old people, dietetic and general hygienic regimes should be even stricter and more closely supervised than in younger persons. In time such patients will succumb to myocardial insufficiency, coronary thrombosis, cerebral insults, or uremia, even when the greatest care has been exercised.
From—"Convalescent Care" (New York Academy of Medicine).

CASE REPORTS

GAS BACILLUS INFECTION OF THE ABDOMINAL WALL IN APPENDICITIS*

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INFECTIONS of surgical wounds by anaerobic gas-forming bacteria are comparatively rare. Infections of this type may occur after any intra-abdominal surgical procedure, but they are most often seen after operations for the relief of intestinal obstruction and of suppurative appendicitis.

Winter[1] was the first man to report what he termed emphysema of the abdominal wall as a complication after laparotomy. He described two cases in 1889.

In 1897, Russell[2] reported two cases which developed on the gynecological service at the Johns Hopkin Hospital.

Simonds[3] in 1915, while working at the Rockefeller Institute, found the *Bacillus welchii*, in a sporulating form, in the pus of most appendiceal abscesses, but he also found these organisms to be present in 90 per cent of normal appendices removed during routine autopsies.

Johnson[4] in 1936, stated that Millar, in 1932, had found but forty-eight reported cases of gas gangrene of the abdominal wall in the literature. To these, he added six additional cases, two being reported from his own work.

It is now a well known fact that the pathogenic anaerobes, together with the colon bacilli, are normally present in the intestinal tract of man. It is not altogether surprising, therefore, that one does encounter an occasional case of gas gangrene of the abdominal wall after surgical procedures attacking the appendix, small intestine and colon. It is surprising that this complication does not develop more often.

The manifestations of this devastating clinical complication which strikes with such vehemence, may well be reported in order that the practicing surgeon may ever be on the alert.

CASE REPORT

A moderately obese white female, 32 years of age, developed acute appendicitis while visiting in a near-by city. When she was seen, one week after the initial attack, the diagnosis was confirmed. She was subjected to surgery one week later, having been free from fever for eight days.

The surgical findings were a subacutely inflamed appendix of the retrocecal type. The cecum presented mild edema and a moderate degree of induration. It was necessary to amputate the appendix at its cecal end first, and liberate the embedded appendix by sharp dissection. The cecal wall was folded over with mattress sutures in an attempt to reinforce the area of perforation in the cecum near the point where the appendix had been removed. Vestiges of a pericecal abscess were found and a Penrose drain was placed in this region.

The pathologic diagnosis was healing acute appendicitis. There are thick fibrous callous areas in the subserosa, which showed focal areas of active suppuration.

The postoperative course was typical of a gas gangrene complication. The temperature was normal before operation; twelve hours later it had risen to 103 degrees and the pulse was 130. Twenty-four hours after operation, the temperature registered 104 and the pulse was 136. Thirty-six hours after surgery, the patient became quite jaundiced, the pulse was 140 and she was profoundly toxic. Inspection of the abdomen showed moderate distention and there was a slight amount of brownish drainage with a mouse-like odor. The skin of the abdomen was

* From the Surgical Service, St. Vincent's Hospital, Los Angeles.

bronze in color. Smears and cultures showed the Welch bacillus. Two doses of anti-gas gangrene serum were given, but the patient expired fifty-hours after operation.

Upon incising the deep layer of the subcutaneous tissues over the right rectus muscle at post-mortem, a vesicular tissue was encountered from which a considerable quantity of a gray, opaque fluid escaped. There were definite globules of fat floating in the fluid and the tissue was crepitant with definite gas bubbles apparent. The anterior surface of the left rectus fascia, in about the mid-part, was entirely defective. The muscle tissue had a gray color, a boiled appearance, a friable consistency. There was definite liquefaction and dissolution of its substance. The same change was noted in the right rectus muscle, with the site of maximum involvement extending along the surgical incision. The change bilaterally ended rather abruptly at the level of the upper transverse tendonous inscription. The process extended laterally into the fibers of the transversalis and internal oblique. This was most marked on the left. The subcutaneous tissues under the discoloration of the skin of the left abdomen were especially indurated, showing diffuse cellulitis. The inflammatory process extended inferiorly in the abdominal wall to the pubis, involving the pyramidalis muscles as well as the recti. The tissues of the labia were swollen and indurated and contained an abundance of fluid.

Microscopic sections of both the right and the left rectus muscles showed a diffuse acute phlegmonous process. The interstitial tissue had been invaded by the exudative product of acute inflammation, and this, together with gas produced by micro-organisms, had tended to separate the individual muscle cells and caused necrosis of large numbers of them. The necrotic cells showed a fragmentation and clumping of the sarcoplasm and polymorphonuclears were visible within many of these muscle fibers. Elsewhere were large areas in which the cellular outlines had been completely lost. At the periphery, the cells were swollen and showed earlier evidences of degeneration. The picture was one of extremely acute cellulitis in the early stages of development. There still remained areas in which myriads of Gram-positive micro-organisms, mostly bacilli, were visible. Several of the smaller blood vessels within the limits of the acute process had been completely thrombosed.

SUMMARY AND CONCLUSIONS

This case presents a rare and fatal complication following appendectomy. Following several attacks of pain in the abdomen, considered to be due to appendicitis, an acutely inflamed appendix was removed and the stump was inverted and reinforced with mattress sutures. It was noted at the time of the operation, that the immediately adjacent area of the cecum and the base of the appendix were considerably edematous and indurated. Almost immediately following the operation, a marked reaction was observed, characterized by a high fever, restlessness and pain in the right labium and in the left side of the abdominal wall. On the third postoperative day, the patient died.

Necropsy revealed an acute, generalized peritonitis and a very extensive gas bacillus infection of the abdominal wall. Apparently the source of the infection was the appendiceal stump, for in this area, a pericecal abscess was found and the tissues of the inverted stump were necrotic.

Two points of rather unusual interest in this case are, first, the fact that the infection of the right rectus muscle was limited in its extension upward by one of the tendonous inscriptions, and secondly, it is curious how the infection could spread from the right rectus across the barrier of the linea alba to involve the left rectus muscle and the left abdominal wall. It is only speculative, but probable, that the infection followed a route via the pyramidalis muscle.

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SARCOMA OF THE STOMACH

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SURGICAL literature has numerous case reports and, indeed, fairly lengthy series of sarcomata of the stomach. In spite of this, gastric sarcoma must be considered very rare. It is also very infrequently diagnosed preoperatively. It is of the greatest importance to make such a diagnosis, since a fairly large sarcoma may still be operable, and those which are not may be materially improved by *x*-ray therapy. Diagnosis is, therefore, of the utmost value. While this is very difficult in many cases, it may be accomplished frequently by giving attention to certain details. The author wishes to report a case diagnosed before surgery; also some points relative to the differential diagnosis are discussed.

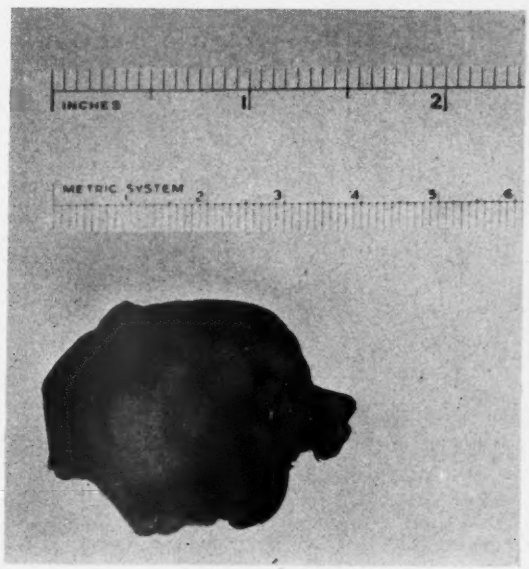

FIG. 1. Illustration of gross specimen. Note the round, dark, ulcerated area which has the appearance of a dimple, and which held the fleck of barium suggestive of sarcoma.

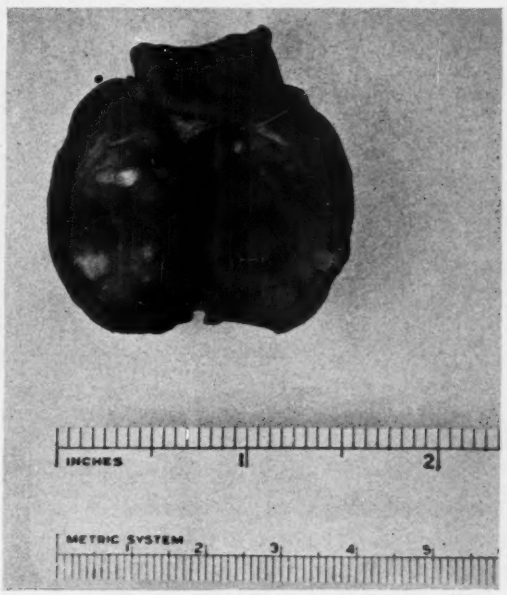

FIG. 2. Gross specimen divided. Note its mottled appearance.

Pathology. About 1 per cent of tumors of the stomach are sarcomas. There is considerable confusion in classification since many are mixed celled varieties. There may be difficulty in differentiating between carcinoma and sarcoma grossly and even microscopically. Cases of carcinoma with metastasis have exhibited cells which resemble sarcoma and have been reported as a mixture of the two. It is believed that any definitely malignant ulcer should be regarded as carcinoma.

Ewing Classification. (1) spindle cell myosarcoma; (2) lymphosarcoma; (3) miscellaneous round cell or alveolar sarcoma.

Spindle cell myosarcomas form solid tumors which may protrude into the stomach or extragastrically; they may remain interstitial; often ulceration occurs; they may grow to tremendous size. Brodowsky's tumor grew to the size of a child's head. In Cantwell's case it weighed 12 pounds, while Baldy's nearly filled the abdomen. Most of these tumors arise from the curvatures.

Lymphomatous tumors occur: (a) as part of the leucemic process; (b) in gastrointestinal leucemia; (c) as part of a general lymphosarcoma; (d) localized lymphosarcoma.

Miscellaneous types. Gross forms assumed by sarcoma vary greatly. The spindle-celled or mixed variety may form a ball-shaped endogastric mass which has the appearance grossly of a benign tumor. However, if there is an ulcerated area in the center which can be seen as a "button filled with barium" on roentgenologic examination, then it marks it more definitely as a sarcoma. The stomach has normal rugae up to the base, and frequently the mucosa is flattened out over the mass.

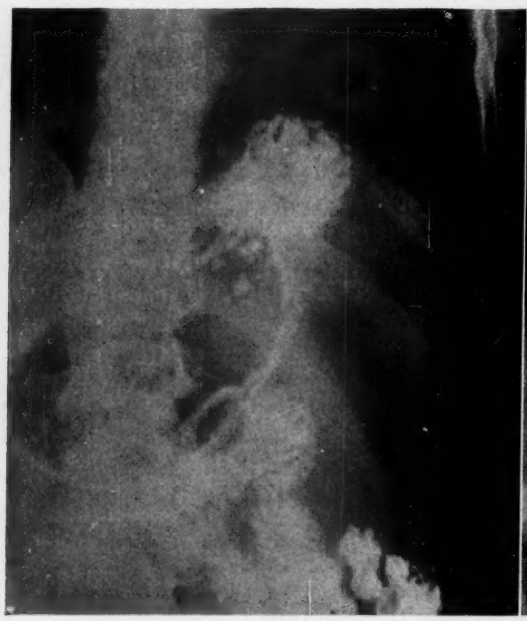

FIG. 3. Sarcoma of the stomach. Note the persistent fleck of barium filling the ulcerated area of the sarcoma.

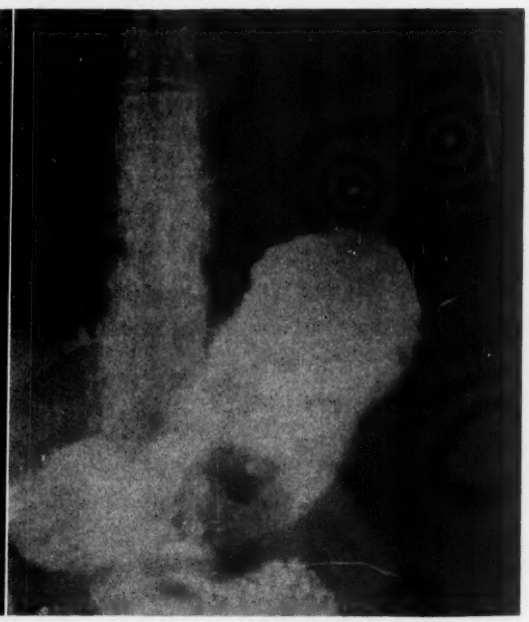

FIG. 4. Sarcoma of the stomach. Note the persistent fleck of barium filling the ulcerated area of the sarcoma.

Sarcoma also frequently extends extragastrically, and may form a dumbbell (as shown by *x*-ray) when ulceration occurs.

Lymphosarcoma begins usually in the submucosa, and may extend widely.

Differential Diagnosis. A careful history is necessary. Bleeding is frequent and often pain is excessive. The patient may be quite well nourished.

A massive tumor leads one to suspect sarcoma. Also, as in the author's case, a deformity which has an ulcerated spot in the center, filled with barium, makes a diagnosis relatively positive.

X-ray is probably the best clinical adjunct. In this connection, the use of a thin barium mixture is advisable. Also the partly filled stomach may yield better results. One must note particularly the rugae, the flexibility of the walls and the peristalsis.

The writer wishes to refer to an article by R. A. Carter and R. Laing, in which this phase of the subject is admirably covered.

The following entities are normally considered: (1) benign growths; (2) benign ulcers; (3) carcinoma; (4) syphilis; (5) lymphoblastic sarcomata; (6) mixed-celled and spindle-celled sarcoma.

Benign tumors are more common than was thought. The incidence now is considered to be from 11 to 17 per cent. They present the following characteristics: (a) usually multiple; (b) 69 per cent are near the pylorus; (c) frequently pedunculated; (d) may form a cluster and be so close together that thick barium may not penetrate to the base—therefore best examined with thin barium with a partially filled stomach; (e) anacidity may be present; (f) hemorrhage may occur in varying degree; (g) symptoms, usually vague, but may be acute should prolapse occur through the pylorus; (h) rugae are normal up to the

tumor; (i) no contracture of the stomach occurs; (j) peristalsis normal; (k) usually are much smaller than the larger rounded endogastric masses of fibrosarcoma, which

Fig. 5. *X*-ray of the same stomach showing the stomach well filled with barium. This does not permit the showing of the outline of the tumor, and particularly the ulcerated area on the tumor. It is, therefore, necessary to take several *x*-ray pictures.

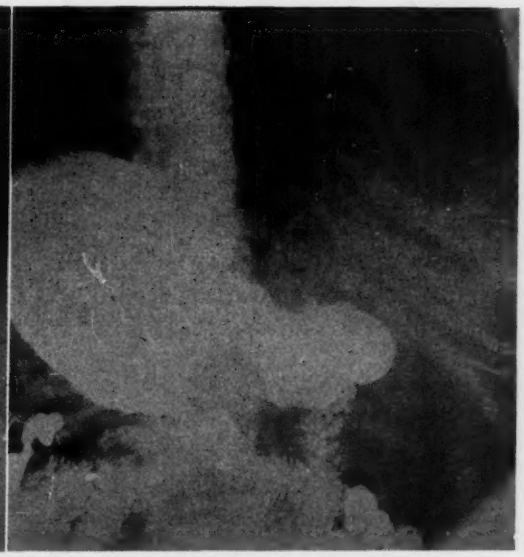

Fig. 6. Cancer of the stomach. (Of interest for differential diagnosis.)

do not exhibit the barium filled "button"; (l) usually occupy the anterior or posterior wall, leaving the curvatures free and do not interfere with peristalsis.

Benign ulcers have a history of dyspepsia and hyperacidity with pain which usually has a definite relationship to food. *X*-ray examination shows deformity, ulceration, and interference with peristalsis; often hypermotility and frequently six-hour retention and spasm.

Where a definite large, flat ulcer occurs, one must rule out sarcoma, and then differentiate between benign ulcer and carcinoma.

Carcinoma. Sarcomas are usually diagnosed as carcinoma since the symptoms are very similar. They have in common, dyspepsia, anorexia, loss of weight, anacidity, and occult blood. However, the following points may be of value in differentiation:

1. Rapidity of growth—greater in carcinoma. Sarcoma may take several years to mature.

2. Age incidence—average age in carcinoma is 61, in sarcoma, 40.

3. Sex—sarcoma is much more frequent in males (70 per cent) than in females.

4. Pain—may be absent in carcinoma and is frequently severe in sarcoma.

5. Gastric hemorrhage is more likely in sarcoma, although occult blood is present in both.

6. Size—the occasional "bulk" of sarcoma may give a suspicion of its character.

7. Any large ulcer should be regarded as carcinoma rather than sarcoma. (Confusion has occurred where mixed types were reported but metastases were carcinoma.)

8. Loss of weight—more frequently occurs in carcinoma. Some stress the well preserved condition of the patient.

9. The presence of a rounded tumor with a barium filled "button" is very diagnostic of sarcoma.

Syphilis. Here there is usually no mass which one can feel by palpation, and the patient's general condition is usually much better than one would expect from the extent of the lesion in the stomach. Serologic examination may also give a clue.

X-ray examination shows: (a) lessened flexibility and motility; (b) lumen of the stomach may be contracted; (c) absence of peristalsis may be marked; (d) pylorus

tends to gap; (e) smooth annular constriction sometimes occurs.

Prognosis in Sarcoma. Balfour states

FIG. 7. The above is an *x*-ray of large multiple ulcers of the stomach. (Of interest for differential diagnosis.)

that of fifty-four cases, thirty-eight were operable. Of this series, two were diagnosed as sarcoma previous to operation. Twelve were alive, the average postoperative period being five years.

Operation. The most logical operation is a wide, partial gastrectomy. However, in certain selected cases, the less radical local resections may be all that is necessary. Rancohoff and Dickson believe this to be true.

CASE REPORT

Male, age 52, weight 140, height 5 feet 6 inches. Occupation: mail clerk.

The patient had had no serious illness. A hernia had been treated successfully by the injection method.

He had been entirely well until a year before when he began having obscure aches and pains in the abdomen. These were not serious in character, and he did not present himself for diagnosis until he suffered a severe hemorrhage and was sent to the hospital. A blood transfusion was given. Having recovered, the patient was *x*-rayed, and the presence of a large, smoothly rounded filling defect, occupying the middle third of the stomach, exhibiting a central fleck of barium was reported. The lesion did not appear to be invading the stomach wall, and did not move or change position.

A recheck examination two days later reaffirmed the findings. A diagnosis of sarcoma of the stomach was made.

Gastric analysis showed blood 3 plus; total acidity of 60, free acid of 45 and combined acid.

At operation no sign of any local extension was seen. No glands were involved, and the liver appeared normal. The tumor was about the size of a tangerine, and was very benign in appearance.

The rugae over the tumor had completely disappeared, the mucous membrane being smooth, but the rugae on the stomach were normal up to the edge of the tumor. The mass was freely movable, showing that there was no infiltration into the gastric wall, and there was little extension along the wall. The ulcerated area which had been seen by *x*-ray marked the mass as a sarcoma. Wide local resection of the tumor was done, and the stomach wall repaired. The patient made an uninterrupted recovery.

Pathologic Report (Brem, Zeiler and Hammack). The specimen consisted of a piece of gastric wall which measured 6 cm. in greatest length. There was a normal-shaped mass 4.5 by 3 by 3 cm. beneath the mucosal surface and grossly demarcated from it. The overlying mucosa showed a small, oval-shaped, depressed area over the midportion of the mass, 1 cm. in greatest diameter and 0.5 cm. in depth. The mass appeared to be continuous with the underlying muscularis.

Section showed a circumscribed tumor just beneath the submucosa and sharply demarcated from it. The mucosa itself had relatively normal glands with some pressure atrophy, while the tumor was very cellular, highly anaplastic and mesoblastic. There was marked pleomorphism of the cells, many of which were spindle, others polyhedral, still others round. They varied greatly in size, some being relatively small and others quite large giant cells with multinuclei. There was an appreciable amount of intercellular stroma. Many of the nuclei were hyperchromatic and there was an equal number of mitotic figures. Alveolar arrangement was missing. A moderate infiltration of the interstitial spaces with round cells and some free blood were noted, the latter possibly of traumatic origin. The blood vessels were relatively

few in number. Pathologic diagnosis was mixed cell sarcoma of the stomach. The patient resumed his occupation six weeks following operation. Five weeks later he had no indigestion, ate a general diet, and had gained 6 pounds in weight. Gastric analysis showed free hydrochloric acid, combined acid, occult blood.

SUMMARY

1. A case of sarcoma of the stomach diagnosed preoperatively is reported.

2. The importance of diagnosis is emphasized since failure may lead to the faulty conclusion that an inoperable carcinoma exists. Operation or radiation may thus be withheld.

3. Operability is greater, and the prognosis better, than in carcinoma.

4. Points of differential diagnoses are given.

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CONGENITAL PYLORIC STENOSIS IN A PREMATURE INFANT FOLLOWED BY GANGRENOUS APPENDICITIS

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GALLAGHER of Philadelphia, in 1937, reported the case of a thirty-one day old infant weighing 3 pounds 8¾ ounces successfully undergoing a Rammstedt operation for pyloric stenosis. The infant was premature, having been delivered by cesarean section from a toxemic negress. Gallagher considered this the smallest baby which had withstood this surgical procedure.

We wish to report a similar case in an infant weighing 3 pounds 12 ounces. Because the baby survived a second major surgical procedure eight months later for the relief of a ruptured, gangrenous appendix, the case is of special interest.

On November 30, 1937, Mrs. A. D. was delivered of a 4 pound male infant under pudendal block anesthesia after spontaneous rupture of the fetal membranes. The gestation period was calculated to be thirty-one weeks (seven and three-fourths lunar months). The mother was unable to nurse the infant and he was given a formula of cow's milk and dextrimaltose. In three weeks he was slightly above his birth weight and was discharged from the hospital.

One week later, a month after birth, the baby was returned to the hospital because of vomiting, loss of weight, and constipation. Although the baby had been vomiting only twenty-four hours, yet its admission weight was only 3 pounds 12 ounces. The baby appeared cyanotic and very dehydrated. Its cry was weak, and only occasionally did it move or kick. All feedings were promptly returned. The general examination was entirely negative. There was no palpable abdominal tumor, nor was peristalsis visible. In view of the history and the lack of physical findings, a diagnosis of congenital pyloric stenosis was made and immediate operation undertaken.

An incision 4 cm. in length was made in the right upper rectus region after infiltration with 1 per cent novocaine. A greatly thickened pyloric muscle was brought into view. It had the typical glistening appearance and extended from the duodenal juncture about 2 cm. into the stomach. The musculature was incised down to the submucosa. No attempt was made to control bleeding. The stomach was replaced and the abdomen closed in layers with fine silk as speedily as possible. The baby was immediately fed small amounts of a cow's milk and dextrimaltose formula which it retained in part. The baby's weight dropped, little by little, until on the sixth postoperative day it was 3 pounds 6 ounces. Semisolid feedings were not used, the baby being kept on the cow's milk formula. However, from the sixth day on the baby made a steady improvement in weight. The wound broke down on the tenth day and complicated the convalescence, but under conservative treatment and adhesive strapping it was completely healed on the thirtieth day. The baby was discharged on the seventieth day weighing 6 pounds 12 ounces.

The infant continued to gain weight at home and remained healthy for a period of five months. On August 27, 1938 the mother consulted a physician because the infant had eaten poorly for the preceding four days and had cried a great deal. Within the next twenty-four hours the baby began to vomit and his temperature rose to 104 degrees. He appeared acutely ill, lying pale and motionless and having shallow, grunty respirations. He cried weakly when moved. The abdomen was distended, rigid, tense, and tympanitic. There was a soft, well defined, tender, lemon-sized mass in the right lower quadrant which could be felt on rectal examination.

The baby was hospitalized immediately. A flat abdominal plate showed distended loops of small bowel suggestive of intestinal obstruction or paralytic ileus. There was a radioopacity corresponding to the mass in the right lower quadrant. A diagnosis of appendiceal abscess was made. Under 1 per cent novocaine anesthesia, a 4 cm. muscle-splitting incision

was made in the right lower quadrant. About 30 c.c. of foul, thick pus welled out. The appendix was delivered from a position deep in the right pelvis and was swollen, tense, necrotic, and perforated near its base. There was considerable plastic exudate on the cecum and terminal ileum. The appendix was amputated and the stump simply ligated. Five cigarette drains were distributed in the pelvis and right iliac fossa. A few black silk sutures were passed to effect a loose closure.

Postoperatively the infant was given subcutaneous infusions of saline and a transfusion of citrated blood. Drainage from the wound was profuse after the third day. On the thirtieth day the wound was completely and solidly healed and the baby was discharged weighing 12 pounds 2 ounces.

At 15 months of age, the child weighed slightly over 18 pounds.

SUMMARY

We report the case of an infant weighing 3 pounds 12 ounces successfully undergoing the Rammstedt operation for congenital pyloric stenosis and later at the age of 8 months recovering from an acute appendicitis with abscess formation.

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CHRONIC RECURRENT JEJUNOGASTRIC INTUSSUSCEPTION THROUGH A GASTROENTEROSTOMY STOMA*

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RETROGRADE intussusception of the jejunum into the stomach is an uncommon sequel following gastroenterostomy. Steher[1] described a case of this type in 1917 and from that time to June 1937 we have been able to find forty-three similar instances described in the literature.

Two clinical groups have been observed. In one group the patients have a brief illness evidenced by epigastric pain and vomiting, first of food, then of bile, and finally of blood. An epigastric tumor may be palpated above and to the left of the umbilicus. Visible peristalsis and upper abdominal rigidity are often present. The usual preoperative diagnosis have been high intestinal obstruction, a ruptured viscus, or an "acute surgical abdomen." In this group the prognosis is grave and surgical intervention should be immediate.

The other group of patients is composed of those who maintain a state of chronic invalidism following a gastroenterostomy for peptic ulcer. The characteristic symptoms, beginning at any time from five days to fourteen or more years after operation, are epigastric pain, nausea and vomiting, which occur at irregular intervals and subside spontaneously. In these patients, as in the following case report, the radiographic examination aids in establishing the diagnosis.

The patient was a white female of 39, with a long history of recurrent attacks of vomiting following a posterior gastroenterostomy which was done for a duodenal ulcer. The radiographic and gastroscopic examinations diagnosed a recurrent jejunogastric intussusception.

She was admitted to the service of Dr. Eldridge L. Eliason in March 1936, with a chief complaint of nausea and vomiting for four days. She had been well until 1919, when she developed a burning pain in the epigastrium. In 1921 an appendectomy was done at another hospital, without relief of her symptoms. In 1926 the epigastric pain and nausea became more severe during gestation, which was terminated prematurely because of nephritis. A second pregnancy resulted in the delivery of a full term child in January 1928. The epigastric pain continued increasing in severity and, in January 1929, a posterior gastroenterostomy was done at another hospital. The patient had an uneventful convalescence but, during the summer of 1929, had a brief spell of vomiting. In March 1936, another attack of vomiting was associated with epigastric pain. Since then she had had recurrent attacks of vomiting. On March 24, 1936, she began to have violent epigastric pain, nausea, vomiting, chills, and headache which lasted, with brief periods of remission, until the time she was admitted to the hospital on March 27, 1936.

The diagnosis on admission was gall-bladder disease. The blood count showed 4,100,000 red blood cells, 77 per cent hemoglobin, 4,700 white blood cells, with a normal differential. The examination of the urine showed nothing abnormal. The Kolmer and Kahn tests were negative. Blood urea nitrogen was 11 mg. per 100 c.c.; blood sugar, 60 mg.; and phenolsulphonephthalein excretion 35 per cent in the first hour and 20 per cent in the second hour. Gastric analysis showed no free acid.

The fluoroscopic examination of the stomach showed a pliable, partially movable, filling defect, within the lumen of the stomach. (Fig. 1.) This defect showed a constant pattern, consisting of parallel curved lines suggestive of mucous membrane folds of the small intestine. (Fig. 2.) There was delayed gastric emptying and barium leaving the stomach by way of the duodenum reëntered the stomach. A diagnosis of intussusception of the small intestine through a gastroenterostomy stoma was made.

* From the Department of Radiology of the Hospital of the University of Pennsylvania.

On April 3, 1936, a gastroscopic examination by Dr. Gabriel Tucker showed a normal cardia, but the pyloric end of the stomach was obstructed by a mass which projected into the gastric lumen. The mass was covered with mucous membrane and had the appearance of the inside of the bowel. On April 6, 1936, at another gastroscopy what previously had appeared as a rounded mass covered by pink mucous membrane was now a thin, flat fold of the same color.

Fig. 1. The large filling defect is seen in the pyloric region of the stomach. This film was made with pressure over the stomach.

Fig. 2. Note the appearance after removal of the pressure. The concentric folds produced by the mucous membrane pattern within the stomach are clearly defined.

On April 13, 1936, a laparotomy was performed by Dr. Eliason. When the peritoneum was opened, a number of adhesions were separated from the anterior abdominal wall. The duodenum was adherent to the wall of the gall-bladder and the under-surface of the liver, but no ulcer could be found. The stomach and transverse colon were then lifted up and it was found that a posterior gastroenterostomy of the isoperistaltic type had previously been performed. There was no evidence of a herniation of the jejunum through the gastrojejunostomy stoma. This stoma easily admitted three fingers. There was an adhesion involving the distal loop of the jejunum in such a way that it tended to invert the jejunum into the stomach. This was divided, permitting the jejunum to straighten out. There was no evidence of a polyp or other tumor in the upper small intestine. The abdomen was closed and the patient left the hospital on April 30, 1936, after an uneventful convalescence.

The patient has been followed in the gastrointestinal department and has had subsequent occasional vomiting attacks. Therapy was given for low plasma protein which seemed to have arrested the vomiting attacks when the patient was last seen in March of 1937.

The evidence is quite conclusive that the patient had a retrograde intussusception of the jejunum which spontaneously reduced itself. The clinical history suggested that this may have occurred many times before the lesion was found, and it is entirely possible that similar episodes have occurred since her last operation, although subsequent Roentgen examinations have not shown a recurrence of the intussusception.

The cause of the recurrent intussusception was not determined definitely, although the adhesion mentioned above was so situated that it might easily have started the process. The type of gastroenterostomy and size of the stoma seem to be unrelated to the occurrence of intussusception. Re-

verse peritalsis, vomiting or some form of local irritation in the jejunum has been considered as an etiologic factor. Some cases, such as those reported by Gutmann and Jobin,[2] are associated with marginitis, ulcer, or malignancy of the jejunum or of the gastroenterostomy stoma.

The roentgenographic signs of intussusception of the jejunum into the stomach in this patient were: (1) the presence within the lumen of the stomach of a partially movable, filling defect, having parallel curved lines simulating the normal pattern of small intestinal folds; (2) the reëntry of some of the barium which had left the stomach through the pylorus by way of the afferent loop and the stoma; and, (3) delayed gastric emptying. Ledoux et al.[3] have also called attention to a dilatation of the stomach and a displacement of the pylorus to the right.

Sibley[4] reported a case which B. R. Kirklin was able to diagnose and reduce under fluoroscopic manipulation. In our patient, the intussusception had evidently reduced itself following the first gastroscopic examination. In some of these patients, multiple examinations or examination during an attack of vomiting, may be necessary to establish the proper diagnosis.

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HYDROPS OF GALL-BLADDER IN A FOUR YEAR OLD CHILD

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DISEASES of the gall-bladder in children have not been reported frequently and a hydrops in early childhood seems to be a rarity.

J. B., 4 years of age, was admitted to the St. Elizabeth Hospital December 3, 1938. The family and past history were essentially negative. Four days before the boy began complaining of a diffuse abdominal pain returning at intervals of half to one hour. The pains lasted only a few seconds and evidently did not affect the well-being of the child who was active as usual. Appetite and defecation remained normal. On the day of admission the child vomited and began crying out with pain at intervals of ten to fifteen minutes, being playful between the attacks.

The physical examination revealed a well nourished child. The tongue was moist, temperature 99.3, pulse 95 of good quality, respirations 20. All physical findings were negative, except for a slight tenderness over the entire abdomen during but not between the spasmodic attacks. The urine was negative. Hemoglobin was 75 per cent; erythrocytes 4,700,000; leucocytes 11,500, neutrophiles 67 per cent, lymphocytes 32 per cent, large mononucleated cells 1 per cent.

The first thought was directed towards an intestinal occlusion by parasites or a foreign body. Although the abdomen was not distended and no repetition of the vomiting occurred, a flat roentgenogram of the abdomen was taken, but furnished no evidence of ileus or a foreign body. Volvulus and intussusception could be excluded in view of the absence of characteristic findings. An infection of the respiratory tract could also be ruled out because there were no subjective or objective chest symptoms and the periodicity of the pains could not be caused by an involvement of the lungs or the pleura. Deep pressure over McBurney's point did not elicit any pain and absence of a localized tenderness or signs of peritoneal irritation eliminated also the diagnosis of Meckel's diverticulitis.

Without a definite diagnosis an expectant treatment consisting of administration of sips of water by mouth and glucose solution by rectum was instituted, but in the course of the next twenty-four hours the condition became aggravated; the temperature remained slightly elevated (99–99.2 degrees) but the pulse rate rose to 130, the child refused drinks and the intensity and frequency of painful attacks greatly increased. A definite tenderness developed in the right upper quadrant of the abdomen and in a lesser degree also in the epigastric region.

Operation was performed under ethylene anesthesia. A right pararectal incision was made with its center at the umbilical line. The appendix was found to be normal and the inspection of other abdominal organs gave negative results with the exception of the gall-bladder which was pear-shaped and very distended, having a diameter of 5 cm. at the fundus—an enormous organ considering the age of the patient. The organ was freely movable and no adhesions or lymphadenopathies could be found. Twenty c.c. of a serous-viscid, nearly colorless fluid was aspirated with a syringe and the cystic and common duct palpated in vain search for a stone. The site of the paracentesis was covered with omentum and the abdomen closed without a drainage. No cholecystostomy was performed because such procedure would offer great difficulties in view of the deep location of the gall-bladder and extreme thinness of the wall and also because it was felt that the condition would subside within a short time. Either a stone in or a cholangitis of the cystic duct must have caused the hydrops. Apparently at the time of the operation the stone was no longer present in the duct and the inflammation of its wall was not intensive as could be judged from the absence of severe reactive signs. Therefore a rapid reëstablishment of the patency of the duct could be expected. The postoperative course confirmed the soundness

of this reasoning: the pains did not recur, the child made an uneventful recovery and was discharged from the hospital ten days after the operation. Two weeks later he was in perfect health.

The examination of the aspirated contents of the gall-bladder revealed a serous-mucoid fluid containing few leucocytes and desquamated cells.

SUMMARY

A case is reported of an acute hydrops of the gall-bladder in a 4 year old male child. Recovery followed an exploratory laparotomy with aspiration of the contents of the greatly distended gall-bladder. The report seems to be justified in view of the rarity of the condition in early childhood.

Most surgeons have seen an occasional backache cured by removal of a chronically infected gall bladder.
From—"The Injured Back and Its Treatment" by Ellis (Charles C. Thomas).

CARCINOMA OF THE AXILLARY TAIL OF THE BREAST

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INTEREST in the axillary tail of the human breast was aroused by two cases of carcinoma of this region which came under the author's care. A brief review of the anatomy of the mammary gland and the citation of these two cases are the basis of this paper.

Commonly breast tissue is rather sharply confined to the circumscribed area in the mammary region. The mammary gland itself is usually described as hemispherical in shape with its flat surface resting upon the fascia covering the pectoralis major and its anterior convex surface in relation with the subcutaneous tissues. Mesially, the breast tissue extends as far as the sternum and laterally to the anterior axillary line; it occupies the area between the third and seventh ribs. There are two or more extensions of breast tissue from the main bulk, however. One is directed downward and inward toward the sternum and the other in which we are more interested, extends upward and outward toward the axilla. This extension toward the axilla is sometimes known as the axillary tail. Under some circumstances, this axillary tail may be entirely separate from the bulk of mammary tissue. These extensions of breast tissue are not to be confused with the condition of polymasia, in which there are supernumerary breasts, each with its own nipple, located upon the chest or abdomen; this latter condition is perhaps a manifestation of man's descent from animal.

The axillary tail of the mammary gland extends upward from the main part of the gland, curves about the border of the pectoralis major toward the axillary space, sometimes terminating just below the axilla. It usually is superficially located and not so well concealed by fatty tissue as is the main bulk of gland. It communicates with the principal mass of breast tissue and drains into the common duct. Rarely this aberrant breast tissue is entirely dissociated from the gland itself and is located beneath the edge of the pectoralis major as a discrete mass.

Obviously the axillary tail of the breast is heir to the same physiologic and pathologic changes as the mammary gland itself. During menstruation it may become enlarged and tender. Likewise, with lactation its cells are stimulated to secretory activity. Its blood supply is less sufficient than that of the breast proper, but the lymphatic channels communicate more directly with the glands of the axilla. In view of the theory that heterotopic tissue is more likely to undergo neoplastic changes than normally placed tissues, one would expect the axillary tail of the breast to show relatively frequent involvement with carcinoma. Such does not appear to be fact, however, if one can judge by the literature and by experience. On the contrary, primary carcinoma of the tail of the breast is quite uncommon.

The symptoms and physical signs of carcinoma of the axillary tail of the breast differ somewhat from those occurring with the usual type of mammary cancer. Discovery of the mass usually takes place at an earlier period on account of the exposed location. This in itself is very helpful in the fight against cancer. The unusual location of the tumor mass may be somewhat confusing when first seen.

It appears to be located too far from the breast itself to be a tumor of breast tissue. The tumor mass is commonly found along the axillary border of the pectoralis major, well up toward the axilla. It may be underneath the edge of the muscle, for the axillary tail occasionally bends underneath the muscle. No connection with the main bulk of breast tissue is evident. In fact, often the

physician does not consider this possibility at all. Just as in breast lesions, pain is uncommon in the earlier stages; tenderness may arise, however, on account of the more exposed location of the tumor. Discharge from the nipple is not to be expected with a tumor located so far from the main ducts. The elevation of the nipple, so frequently seen with primary breast tumors, is uncommon with this lesion because of its relatively distant location. The tumor mass is usually rather hard to the touch; it may be tender. Its mobility will depend upon the duration of the process and the extent of involvement of surrounding tissues. Careful examination of the breast on the affected side is negative as a rule, except possibly for changes of chronic mastitis. Involvement of the neighboring lymphatic glands takes place at an earlier date than with tumors of the breast proper.

Carcinoma of the axillary tail of the breast must be differentiated from tumors arising in the subcutaneous tissue of this region. Benign tumors such as lipoma or fibroma or cysts are very unusual in this location. Ordinarily one has little difficulty in differentiating such tumors from the malignant. Malignant tumors arising from other structures in this area are rare. As lymph glands are seldom found here, tumors of lymphatic origin are uncommon.

In considering treatment of carcinoma of the axillary tail of the breast, one is torn between the conservative and radical viewpoints. Usually local excision of the tumor mass is first done as the essential means of establishing the diagnosis. When the diagnosis is established by frozen sections or later study of the tissues, the problem is to decide if one should proceed with radical amputation of the breast or not. If there does not appear to be evidence of any tumor in the breast, it seems unnecessary to resort to radical measures, providing a wide excision of the primary mass was done. In one of the author's cases, subsequent amputation of the breast was done because of uncertainty of just what was in it. This procedure would seem justified if the breast does show any suspicious findings. If there is any involvement of the axillary nodes, it would seem safer thoroughly to clear the axilla. The value of radiation therapy requires no discussion; postoperative radiation is the best safeguard against the possibility of recurrence.

CASE REPORTS

Case I. C. L., married female, age 59, was referred to me by her physician in December, 1931 because of a "bunch" in the region of the right breast. She stated that she had noticed the mass about seven weeks before; it was not tender or painful; she did not know if it had increased in size; there had been no discharge. This patient had never lactated. The previous history was negative.

Upon examination a mass 2 by 3 cm. was found in the anterior axillary line, well above the right breast. The mass was hard but freely movable. There was no evidence of involvement of the axillary glands. The right breast appeared negative except for some areas of infiltration such as occur in chronic mastitis.

Several days later, local excision of the tumor mass was done under gas and oxygen anesthesia. At operation the mass was found to be well encapsulated and located under the axillary border of the pectoralis major; it was not particularly adherent and there was no evidence of involvement of the surrounding structures. Microscopic study of the mass showed it to be adenocarcinoma arising in breast tissue. The postoperative course of the patient was not remarkable. In view of the report of malignancy and the question about the right breast, this was amputated three weeks later. Careful study of the removed breast with many sections showed no definite evidence of malignancy; the breast did show changes of chronic mastitis, however. Convalescence from this operation was uneventful. This patient has received no further treatment; recent check-up shows her to be free of any sign of recurrence of the tumor.

Case II. W. E., a 50 year old spinster was referred June 7, 1932 complaining of a tumor mass of the lower part of the left axilla. The patient stated that about one year previously she had injured this area, but that she had noticed nothing unusual until three months before coming to her physician. At that time a button-like thickening appeared in the skin overlying the axillary fold on the left side.

The swelling had not been red or inflamed; it did not seem to enlarge; there had been no discharge; there never was any pain.

The previous history was essentially negative. Lactation had not occurred; the menopause had been passed two years before and was uneventful.

Examination showed a hard tumor mass, about 2.5 by 2 cm., located in the anterior axillary fold, just below the axilla. The overlying subcutaneous tissue was moderately adherent but the mass was not adherent to the underlying tissues. No glands were found in the axilla. The left breast seemed negative.

A few days later, under anesthesia with gas and oxygen, the tumor mass was widely excised; the breast and axilla were not disturbed. The patient made an uneventful postoperative recovery. Sections from the mass showed a scirrhous carcinoma arising in breast tissue. Postoperative radiation was suggested but not accepted by the patient. Four months later, there was a questionable mass in the left axilla. In view of this finding, deep therapy was insisted upon and eight treatments were given at weekly intervals with a dosage of 355 R units each time. Under these treatments, the mass in the axilla disappeared.

Recent examination reveals no evidence of recurrence.

CONCLUSIONS

1. Carcinoma of the axillary tail of the breast is unusual and offers some difficulty of diagnosis.

2. Wide local excision of the mass, followed by radiation therapy seems sufficient in the absence of evidence of involvement of the breast.

RUPTURED AORTIC ANEURYSM, SIMULATING RENAL TUMOR*

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ALTHOUGH aneurysm of the abdominal aorta is regarded as comparatively rare, a review of the literature would tend to disprove this impression. Since many cases are not autopsied, and a great proportion of those that are do not find their way into print, it can readily be appreciated that the incidence is far greater than is commonly believed.

McGeachey and Paullin report over 500 cases of ruptured aneurysm in which the diagnosis was made ante-mortem in only nine instances, and they add three correctly diagnosed cases of their own. More discouraging than the failure to recognize the lesion have been the surgical results aimed at its correction.

Perforation of an abdominal aortic aneurysm in a male of 47, simulating a retroperitoneal or renal neoplasm presented an interesting diagnostic problem. Further, our experience with the two common terminal complications, namely, perforation and rupture, prompted this presentation.

CASE REPORT

A white male, 47 years of age, was admitted to the St. Mary's Hospital, Passaic, New Jersey, on November 5, 1937. He had severe steady pains in the left lumbar region, radiating to the pelvis and accompanied by nausea and vomiting.

Following a high diving stunt twenty-six years before he suffered pain in the left lumbar region which confined him to bed for several days. He felt well after that until five years before admission, when he again experienced an attack of pain in the left side. After a urologic survey in a Brooklyn hospital, he was informed that an enlarged calcified gland pressing on the kidney was responsible for his pain. His last attack immediately prior to admission was so severe, causing shock and collapse, that immediate hospitalization was advised.

The left eye had been enucleated when the patient was 15 years old, due to an injury sustained when he was 9. Five years before admission he was treated at the Neurological Institute for a paralysis which was attributed to chemical poisoning.

Physical examination on admission revealed a well-nourished, adult male, who was pale, cold and clammy, and apparently recovering from shock. The tongue was dry. A systolic and diastolic murmur was heard at the cardiac apex. The diastolic murmur was transmitted to the axilla, and up into the neck. A diastolic murmur was also heard over the aortic area. The abdomen was tense along the entire left side, and a large tumor mass was palpated in the left lumbar region, this mass being slightly tender, and semi-solid in consistency. The reflexes were present and active.

Shock therapy was instituted. Three days after admission, a urologic consultation and survey was requested by the surgical staff, as a renal neoplasm was suspected. A scout roentgenogram showed a horn-like projection about 1¼ by 1 inch calcified, and apparently overlying the second lumbar vertebra, extending out to the lower border of the twelfth rib. Calcification was also noted along the outer margin of the psoas muscle, extending downward in a linear, streaky fashion. While the right psoas border was sharply delineated, the left was completely obliterated, and there was no apparent curvature of the spine. At the time of cystoscopy, ecchymotic areas were plainly visible in the left lumbar region, and over the dorsum penis. The bladder efflux was grossly clear, the mucosa and ureteral orifices were normal in appearance. Indigo-carmine intravenously appeared from the right ureter mouth in eighteen minutes, and from the left in twenty-two minutes, the concentration of dye being stronger on the right. A French No. 6

* Read before the Section of Genitourinary Surgery, New York Academy of Medicine, February 15, 1939.

shadowgraph catheter passed up the right ureter for a distance of 27 cm., met no obstruction, and there was no retention, the efflux on this side being grossly clear. The catheter passed similarly on the left, but aspiration yielded 5 c.c. of retained urine. The roentgenogram with the catheters in situ showed a marked displacement laterally of the left catheter. Bilateral pyelography revealed both pelves within normal limits, with the left pelvis showing a slight degree of dilatation. The left pelvis was displaced laterally and upward, so that the upper pole of the left kidney almost reached the border of the ninth rib. The lateral deviation of the ureter led us to believe that we were dealing with a retroperitoneal mass, displacing the kidney and the ureter, and causing pressure at the ureteropelvic outlet.

Fig. 1. Scout roentgenogram showing horn-like calcified process and streak of calcification.

Fig. 2. Bilateral pyelograms showing displacement of kidney and ureter by hematoma, the result of perforation.

The temperature on admission was subnormal, and reached 100 degrees two days later.

Urinalysis showed acid reaction of the urine and a specific gravity of 1.021; there was a faint trace of albumin and a few hyaline casts. Hemoglobin was 70 per cent; the red count was 3,500,000, the white 15,000, with polys 87 per cent, small lymphcytes 7 per cent, eosinophiles 4 per cent and monocytes 2 per cent. The Wassermann was negative. Three days later, the hemoglobin had fallen to 40 per cent and the red cells to 2,500,000: the white cells were 17,000 and polys 81 per cent. The low grade temperature and leucocytosis admitted the possibility of an inflammatory lesion, possibly perinephritic. Needle exploration failed to reveal the presence of pus.

The possibilities further considered in the differential diagnosis were: (1) retroperitoneal neoplasm; (2) sarcoma arising from the lumbar vertebra; (3) an old retroperitoneal hematoma, with calcification; (4) a sacculated abdominal aortic aneurysm with perforation. Sarcoma, in view of the long history and general well-being of the patient, was quickly rejected as a possibility. The signs and symptoms pointed to an internal hemorrhage, probably arising in a perforation of an aortic aneurysm, or from a hemorrhagic retroperitoneal neoplasm.

The following day, an extraperitoneal lumbar incision was made under spinal anesthesia. This approach was selected because of the bulging mass in the left flank. A large hematoma, the size of a fetal head at term, was found displacing the kidney upward and laterally, and undermining the musculature of the lateral wall.

After evacuating the hematoma, we encountered a large aneurysm which appeared to be one and one-half times as large as the kidney, and which seemed to be bleeding from a perforation on its under and lateral surface. There was no pulsation visible.

The bleeding large aneurysmal sac, the displaced kidney, and the depth of the wound made visualization of the neck of the sac extremely difficult, so that it was decided to apply tamponage to the bleeding point, and approach the problem transperitoneally at a later date.

The patient received an 1100 c.c. direct blood transfusion (two donors) during the operative manipulations, and he was returned to his bed in fairly good condition. He did remarkably well, and six days postoperatively the hemoglobin count rose to 75 per cent, the R.B.C. to 3,800,000 and the W.B.C. to 15,000.

On the ninth postoperative day, the patient suddenly developed signs and symptoms of an unusually severe internal hemorrhage, which was ascribed to rupture of the sac. The long duration of the aneurysm led us to feel reasonably certain that collateral circulation was established, and that ligation of the aorta might be attempted. He was, accordingly, taken to the operating room, and explored transperitoneally. The sheath of the aorta was incised, a heavy No. 6 braided silk ligature passed around the aorta and tied just below the renal arteries. The patient died almost immediately afterward. An attempt at transfusion during this procedure was unsuccessful, because of the collapsed condition of the veins.

FIG. 3. Aneurysmal sac with site of rupture.

FIG. 4. Aneurysmal sac opened exposing large calcific masses.

The pertinent findings at autopsy (Dr. Kastler) were:

The pleural cavities contained no free fluid. The lungs were very pale, with small atelectatic areas present in the lower lobe. There was no evidence of infarction or pneumonic infiltration.

The pericardial sac contained the usual amount of clear, straw-colored fluid. The visceral and parietal pericardium were smooth and shiny. The visceral pericardium showed some fibrous thickening. The heart was very much enlarged, and showed all signs of a symmetrical hypertrophy. The heart muscle was very pale, and the valves were thickened. The aorta and coronary vessels showed an arteriosclerotic process.

The most striking finding on opening the peritoneal cavity was a large, extraperitoneal mass that filled practically the entire left side of the abdomen, pushing the spleen and kidney upward and displacing the small intestine to the right. On close inspection, the mass was found to be a large hematoma originating from a huge aneurysm of the descending aorta that had ruptured toward the left. The aneurysm was found to be about the size of a child's head

and had developed at the region of the celiac axis. The descending aorta and sac of the aneurysm showed arteriosclerosis and calcification to a pronounced degree, and the formation of large atheromatous plaques. The aneurysmal sac was devoid of intima and was lined with laminated thrombi. On the left side of the aneurysm a recent rupture about 2 inches long was found. Microscopic examination of the aorta showed diffuse arteriosclerosis. The cause of death: ruptured aneurysm of the descending aorta.

COMMENT

This case emphasizes the fact that aneurysms of the abdominal aorta may exist for lengthy periods and remain relatively asymptomatic, until terminal complications occur. The clinical manifestations, when present, are due for the most part to pressure on neighboring organs. Ureteral obstructions with hydronephrosis, vertebral erosions with root pains or paraplegia, intestinal or gastric obstruction, and portal, splenic or mesenteric vein thrombosis are some of the conditions which may indicate the existence of this lesion.

Syphilis, the cause of most aneurysms, was in this case excluded by the history, and the clinical and serologic findings. The extreme degree of calcification and the sclerotic changes in the aorta and coronary vessels suggested arteriosclerosis as a possible underlying cause. The history of trauma sustained twenty-six years previously appeared highly significant. Injury to the aorta with resulting intramural hemorrhage and fibrosis could result in a site of lowered resistance in the wall of the aorta. The latter, yielding to the high intra-aortic pressure, could very well have resulted in the aneurysm.

Perforation with gradual bleeding into the retroperitoneal tissues, or sudden explosive rupture with extensive hemorrhage resulting in sudden death, are the two terminal complications, both of which occurred in our case.

The long duration of the lesion and the thought that collateral circulation had possibly been established, plus the fact that the patient was certain to bleed to death in a few minutes, were the factors that influenced us in attempting ligation. Ligation of the abdominal aorta carries with it a very high mortality, and although Matas, in his exhaustive chapter on aneurysms in Keen's Surgery, offers the hopeful suggestion that gradual occlusion may solve the problem, this view is not shared by research workers in the field of aneurysmal surgery. It is their opinion, that ligation of the abdominal aorta below the inferior mesenteric artery taxes the heart with so enormous an amount of work that only an absolutely and functionally capable organ outlives the strain and death ultimately ensues from cardiac failure.

According to Matas, the abdominal aorta has been ligated fifteen times since Sir Astley Cooper performed the first operation in 1817. Death followed in each case, though the end was delayed to the forty-eighth day in W. W. Keen's case, and to the thirty-fourth day in Tillaux'. If the patient survives the cardiac strain, which is extremely rare, he still has to face the prospect of sudden death as a result of rupture or ulceration of the aorta at the site of the ligation.

Recent reports in the literature would seem to indicate that this problem, which has baffled the surgical world, is being overcome. I refer particularly to the work of Blakemore and King, who have evolved a method of wiring aneurysms. With this method they are able to determine: (1) the variety of aneurysms; (2) approximately the amount of wire that will be necessary to impede the blood flow; (3) the occurrence of mass clotting of aneurysms if a final segment of wire requires only from 3 to 4 amperes for heating to 80°C. The average amount of wire used in the eleven cases reported was 118 feet. Of these eleven cases, there were four deaths and seven survivals. Two were abdominal aortic aneurysms. Both were wired and heated to 80°C. One patient has been symptom-free for two years, while the other was alive and well eight months postoperatively. They

conclude that a thoroughly clotted aneurysm may be inactivated indefinitely, that their method and technique is safe and efficient that heating wire to 80°C. causes a heat inflammation which promotes adherence and organization of the clot deposited, and that the absence of signs and symptoms of growth and relief of pain in seven living patients operated upon by their method justifies the recommendation of this procedure.

SUMMARY AND CONCLUSIONS

1. This case is reported because of the unusually long duration of an abdominal aortic aneurysm, traumatic in origin.

2. The diagnosis was suspected preoperatively from the clinical history, the urographic and roentgenologic findings, and the presence of a tremendous hematoma occupying the entire retroperitoneal space. The diagnosis was further strengthened by the fact that there was no evidence of lues.

3. Ligation of the abdominal aorta was attempted.

4. The electrothermic coagulation method of treating aneurysms offers greater promise of relief and cure than any of the measures employed heretofore.

5. Earlier diagnosis will afford opportunity for therapy before terminal complications occur.

The writer wishes to express his deep appreciation to Dr. B. Willis, Attending Surgeon, St. Mary's Hospital, Passaic, for permission to report this case, and for his helpful suggestions in the operative and postoperative management.

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NEW INSTRUMENTS

A NEW APPARATUS FOR WELL LEG TRACTION

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THIS apparatus is similar in principle to other types of traction appliances in which the well leg is used to fix and reduce the injured leg, using the pelvis as a fulcrum. It is easy to apply and simple to construct. The cost of assembly is small. It can be built by any carpenter, or even a physician who can use tools fairly well.

Description. The apparatus was first built by one of the authors (M. B.), following a suggestion of Dr. N. A. Carey of Oakland, California. It resembles a trapezium with parallel sides of unequal length, the shorter side at the top. The non-parallel sides are equal in length and converge towards each other from below upward. The corners are hinged. A screw turnbuckle joins opposite corners of the parallel sides. The construction is of wood. (Fig. 1.) At first it was incorporated in the outer layer of padded plaster casts applied to both legs. By the action of the turnbuckle the well leg was pushed upward and the injured leg drawn downward. Later the wooden sides which formerly were incorporated in the casts were made wider and were hollowed out so as to fit the outside of the casts. Four metal posts, two on each side, were added, having a knob at one end and a slot at the other. The ends of the posts were turned back to keep the attached bands from slipping. These metal bands had a number of eye-holes at one end and a wing nut at the other. The length was adjusted by choosing the correct eye-hole passing the band around the cast and tightening the wing nut to the other end of the post. This modification was made to provide greater flexibility by allowing reapplication or additional adjusting without removing the casts. Experience proved that at times readjustment became necessary to obtain satisfactory reduction of the fracture. This was impossible without removing the outer layers of the casts. An added consideration was that the casts became less heavy and cumbersome. The end between the knees later was made adjustable to allow for longer and shorter legs. It was realized that the distance between the knees varied with the length of the legs.

Method of Application. Padded casts are applied to both legs, from the base of the toes to the knees. Starting with stockinet followed by several layers of glazed cotton, the points of greatest pressure are next protected by covering them with ¼ inch felt. These danger points are: on the well leg the heel, the bottom of the foot, and the medial side of the leg and knee; on the injured leg the medial side of the knee and leg, the heel, and the dorsum of the foot. Gauze bandages are wound tightly around the padding to reduce the bulkiness. After placing a moulded plaster splint on the inside of the leg and the bottom of the feet plaster bandages are applied. About six 4-inch plaster bandages are used for each cast. The plaster is allowed to harden for several hours to avoid denting, which might occur if the apparatus were applied immediately before the plaster had set. The distance between the patient's knees is gauged with the apparatus in place and the upper bar is adjusted to meet the distance correctly. The bands are passed around the casts. The closest eye-

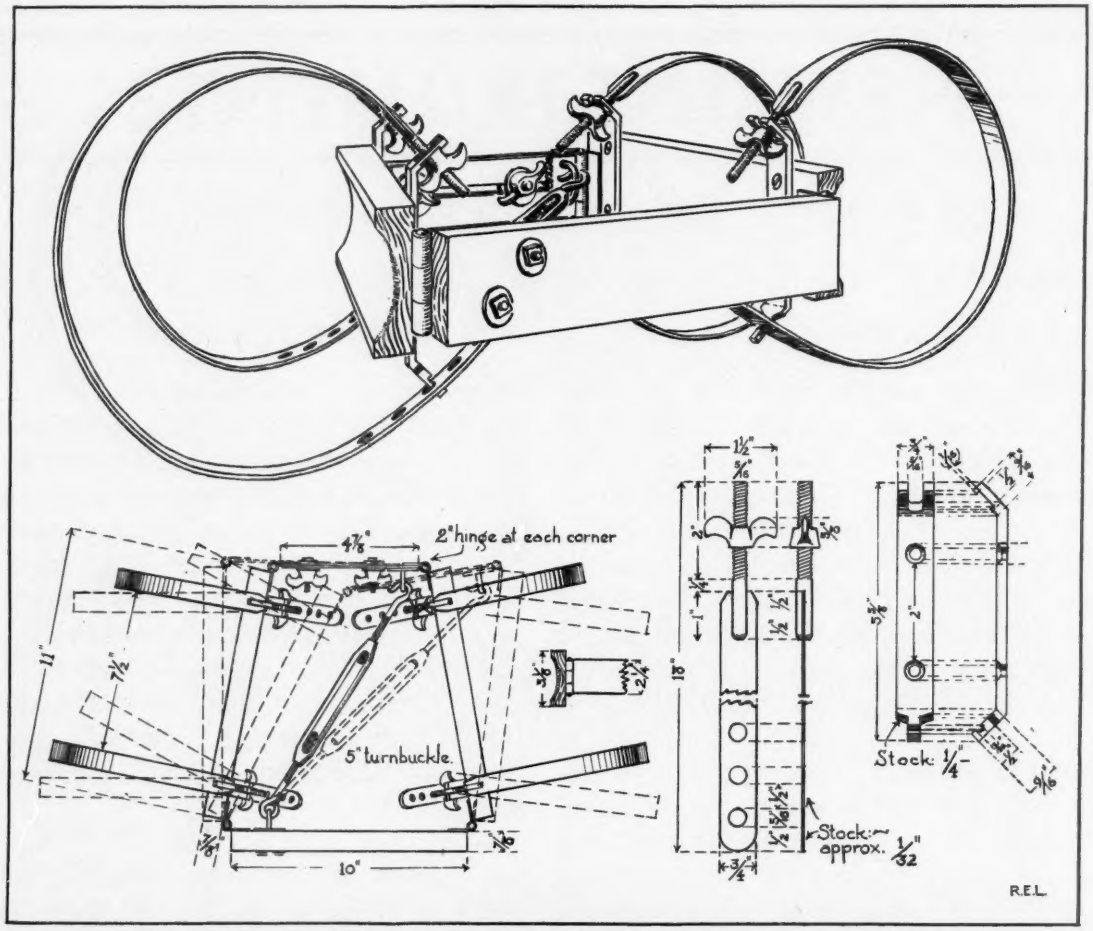

FIG. 1. Apparatus shown above; construction plans and measurements below.

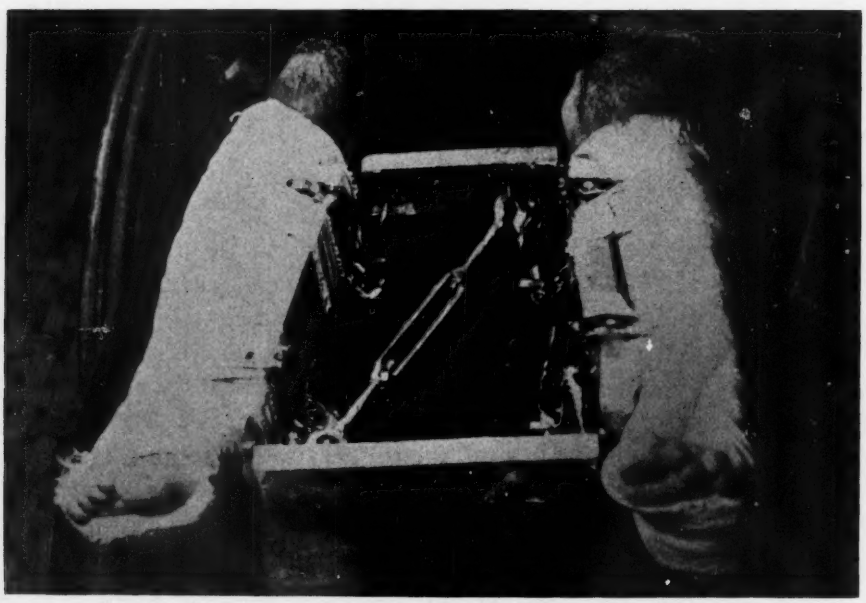

FIG. 2. Showing apparatus in position. Patient in wheelchair.

hole to size is chosen, and the screw nuts tightened to the metal posts. The turnbuckle is now tightened slowly, giving several turns two or three hours apart. Reduction is thus accomplished slowly over a period of ten to twelve hours to avoid producing unnecessary muscle spasm or displacement of the bone fragments. If the patient's condition permits, reduction may also be effected immediately by the Leadbetter maneuver carried out under local anesthesia. In the latter instance the turnbuckle is immediately tightened sufficiently to maintain the reduction. As soon as the fracture is considered satisfactorily reduced, one turn of 2-inch plaster is placed over the bands to keep them from sliding on the cast. This is done because the patients are turned frequently and are also moving about in wheel chairs. The screw bolts also sometimes loosen and allow the bands to slip in spite of careful supervision.

Type of Fracture. The apparatus is used in fractures of the upper end of the femur, both extracapsular and intracapsular. Intertrochanteric fractures unite readily if well reduced. This method is ideal for them. Fractures of the neck of the femur have advocates for operative and nonoperative treatment. If insertion of a pin or nail is not proposed, the results will be as good with this method as with other nonoperative procedures. Over 100 cases have been treated. The treatment differs very little from the general principles of fracture care, so that a detailed discussion is unnecessary. Immobilization is maintained the necessary length of time, depending on the site of the fracture, the age, the general condition of the patient, and the findings in subsequent *x*-rays.

SUMMARY

1. A new apparatus for well leg traction is herewith presented.

2. In over 100 cases the results have been found as satisfactory as with other nonoperative methods.

3. The construction is simple and economical, adapting it to wide usage.

4. Its mode of construction, method of application, and type of case for which suitable, are described.

NEW APPARATUS FOR STORING, FILTERING AND ADMINISTERING BLOOD

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FOR the past year this apparatus has proved extremely satisfactory in the administration of over 4,000 transfusions from the blood banks of Harper Hospital, Grace Hospital and Receiving Hospital, Detroit, Michigan. It permits of a completely airtight system for taking and storing blood that is simple, inexpensive and practically foolproof. After substituting the internal filter cork when ready to administer, the operator can give blood with the same ease as that of glucose infusion. By this airtight method transfusion reactions have become a rare occurrence.

In the diagram, "C" represents the suction apparatus in place ready to draw blood. The 600 cc. bottles are autoclaved containing 70 cc. of 2.5 per cent sodium citrate ready for use. To draw blood, the double needle, "D," of the suction apparatus is pushed through the hole in the metal disc, and on through the small rubber plug, "b," just as one penetrates a rubber-stoppered vial. When the donor needle is in the vein, occasional compression of the rubber bulb provides ample, but never an excessive, amount of vacuum. Occasional twirling of the flask properly mixes the citrate and blood. When sufficient blood has been obtained, the suction apparatus is withdrawn, thus automatically sealing the flask so that at no time is room air able to enter the flask of blood. In the preparation of plasma reserves, this same apparatus is used to withdraw the supernatant plasma from all stored blood reaching seven days old.

"D" illustrates the simple structure of the suction apparatus. A continuous fifteen-gauge needle conveys the blood into the bottle; while an eighteen-gauge needle, which is fused to it, withdraws air from the bottle when the rubber bulb is compressed. Especially constructed ball bearing valves prevent air from entering the bottle.

"E" shows the assembly of the donor bottle closure, "a" being the solid rubber closure cork, while "b" is a soft rubber plug (¼ by ½ inch) which fits snugly into the depression of "a." The metal disc, "c," perforated in the center, holds the plug, "b," firmly in place, while the screw top, "d," fixes the entire closure, ready for use. The plug, "b," is a special vial-type rubber and may be used a number of times before replacing.

"B" represents the internal filter cork, which is put in place just before giving the blood. The filter is reinforced 100 mesh stainless steel, which is easily removed for cleaning, is practically indestructible and never becomes clogged. A glass breather tube permits administration by gravity by a simple intravenous tubing set as shown in "A."

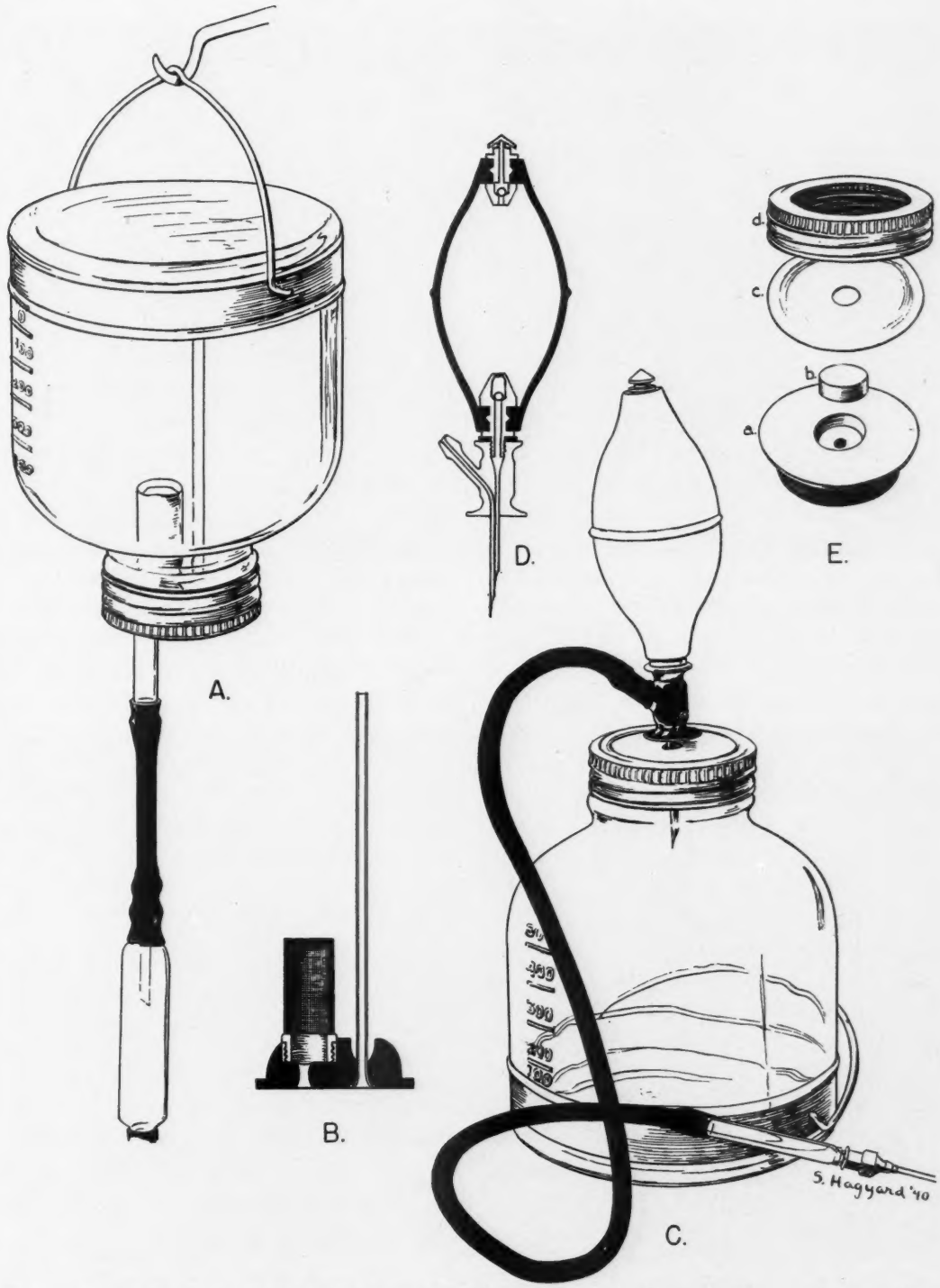

FIG. 1. A, method of administering the blood by gravity; B, internal filter cork assembly; C, suction apparatus in place ready to draw blood from donor or supernatant plasma from stored blood; D, structure of suction apparatus; E, assembly of cork used in taking and storing blood.

A MAGNIFIED OPERATIVE FIELD

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THE use of the binocular loupe for magnification of small objects has been of great advantage to workers in many branches of science. In recent years, a standard model loupe with open sides has been manufactured by various optical firms and has been accepted by those surgeons who at times require magnification of certain structures in their operative fields. This standard loupe projects approximately 4 inches ahead of the face and consists of two small prisms supported on two converging stalks that attach solidly to the bridge of the wearer's regular glasses.

Although this loupe serves to magnify a portion of the operative field, it has several disadvantages: (1) The area enlarged is often too small to be useful in a great many types of work. (2) The lenses are small, so that they occupy only the center of the normal visual field. The constant change in focus from the peripheral to the central field is likely to be disturbing to the wearer if it is necessary to work with these glasses for a long period of time. (3) The supporting structure for the lenses is heavy and, being attached to the bridge, falls in the field of vision. (4) The glasses are not sterile and are likely to be touched by the operator or his assistant. (5) For the stages in the work in which the wearer does not need any magnification it is impossible to get rid of the magnified portion without removing or changing entirely the pair of glasses.

In order to eliminate some of these difficulties, a new type of binocular loupe was designed by the author and built by A. E. Covelle of Boston. A heavy steel frame was fit with the author's corrected lenses. Across the top of this frame a hollow brass rod was soldered with a small platform on each end. Reinforcing side struts were carried down to each ear piece. A tape was attached from the center of this cross-bar to pin to the operator's cap, thereby balancing the weight of the loupe and taking the pressure off the bridge of the nose. This part of the apparatus is unsterile and is put on before the surgeon starts to scrub. (Fig. 1.)

When the stage of operation requiring enlargement is reached, the nurse lifts the second portion of the loupe from a sterilizing solution, dries and attaches it. (Fig. 2.) This second sterile unit is made up of two large magnifying glasses cemented into a solid frame. At one end of this frame are two clips that fit and clamp down tightly into the two end platforms of the first piece. The nurse is able to attach this sterile part to the foundation glasses without contamination of either herself or the enlarging apparatus. The magnifying lenses are on a swivelled bar held in place by side springs so that the operator may turn them up out of the field or down without danger of contamination. (Fig. 3.) Because the lenses are much larger than those in the standard loupe and are butted together in a line corresponding to the exact center of the operator's face, the entire central visual field is magnified. The change from the magnified field to the normal field is then easily made by turning the lenses up out of the line of vision. (Fig. 4.) The supporting structure for this whole apparatus is out of the normal visual field, therefore, does not disturb the wearer. Because the projecting portion is sterile, contamination from touching the loupe is eliminated. When the operator reaches the stage when magnification is no longer needed, he may continue to wear the loupe turned up out of the way,

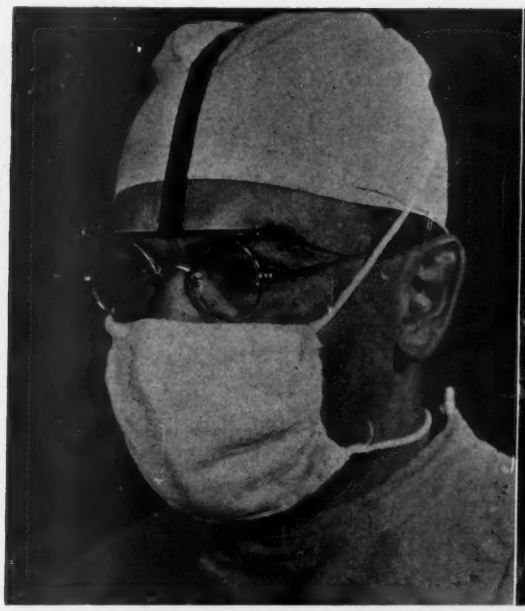

Fig. 1. The basic unsterile unit in place. Note cross-bar with end plates where the junction is made with the sterile second unit.

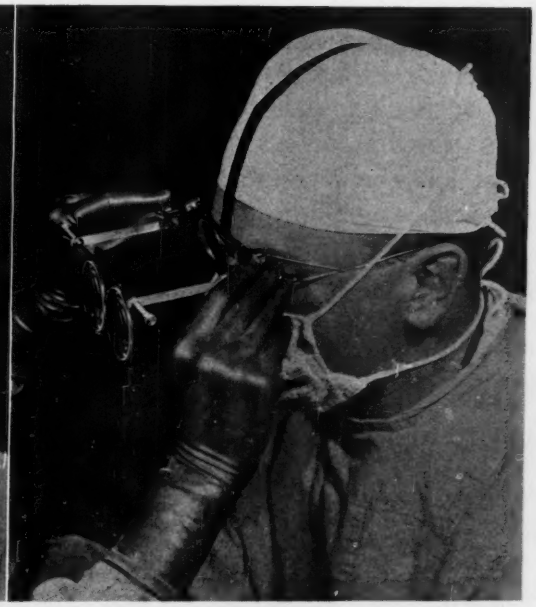

Fig. 2. Nurse applying sterile magnifying apparatus. With care this is done without contamination and does not appear as perilous as in this photograph.

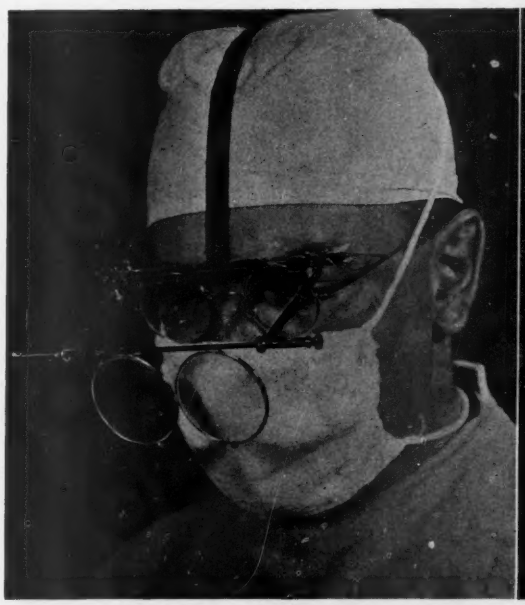

Fig. 3. Entire unit in place for working in a magnified field. Operator, upon raising his eyes, sees directly in front of him through a normal field.

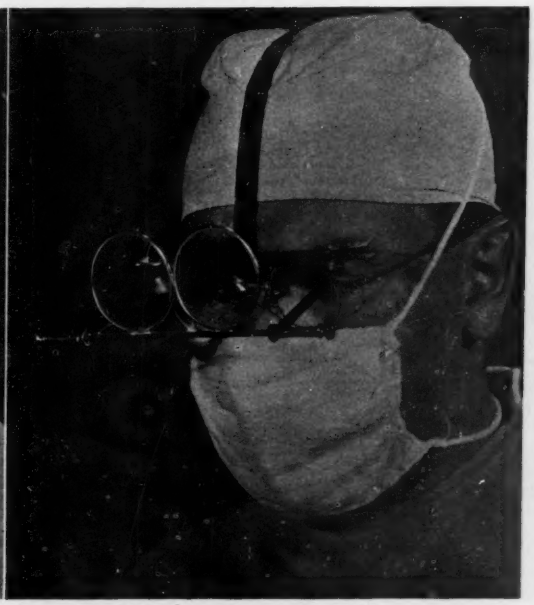

Fig. 4. Magnifying loupe has been turned up by the operator so that the central visual field is now normal. He may turn this down again at will without contamination.

or it may be unclipped by the circulating nurse, leaving the first portion with his normal corrected lenses in place.

In the author's loupe the glasses of the projecting portion consist of two plus four biconcave lenses set forward 2.75 inches from the basic unit. This arrangement produces a depth of reasonably sharp focus, approximately 1 foot. It is, therefore, not necessary for the operator to remain in a fixed position since movement of the head within this 1-foot range does not tend to throw the structures in the operative field out of focus.

As far as can be determined all the disadvantages of the standard loupe have been eliminated with this type of glasses. It was designed principally for use in plastic surgery in which it is frequently necessary to change from the normal to the magnified field for fine, minute work. It has proved so advantageous to the author that it was thought it might be useful to workers in plastic, and other fields of surgery.

THE potentiometer indicator is an instrument which was devised to make direct readings of the skin temperature. It is similar to the dermatherm and consists of a galvanometer and thermocouple arrangement. From—"Peripheral Vascular Diseases" by Kramer (Blakiston).

SPECIAL MONOGRAPH

The Diagnosis and Treatment of Hyperthyroidism and Associated Conditions

BY

ARTHUR GOETSCH, S.B., M.D.

AMERICAN JOURNAL OF SURGERY, Inc.

NEW YORK • MCMXL

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THE DIAGNOSIS AND TREATMENT OF HYPERTHYROIDISM AND ASSOCIATED CONDITIONS*

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BROOKLYN, NEW YORK

GOITER unqualified, is an enlargement of the thyroid gland. Hyperthyroidism or thyrotoxicosis denotes a toxic state due to an excessive amount of thyroid hormone in the circulation. It results from overactivity of the thyroid gland regardless of the fundamental pathology. The specific thyroid hormone, thyroxin, is an immensely potent drug and an exceedingly small fraction of a grain is capable of producing nervous symptoms even in a normal person. When thyroxin accumulates in the circulation in excessive amounts, a high degree of hyperthyroidism is produced; and when diminished amounts are present, varying degrees of underfunctioning or hypothyroidism result. It is likely that the various types of toxic goiter secrete bodies similar to thyroxin since the symptoms produced, while not wholly identical, are similar to those which can be caused experimentally by overdoses of thyroid extract and thyroxin. In dealing with goiter and hyperthyroidism, therefore, we need not discuss the matter of a so-called "dysthyroidism," due to an altered secretion, but keep in mind the probability of toxic bodies analogous chemically and pharmacologically to the pure crystalline thyroxin first isolated by Kendall. It follows obviously that treatment is designed to abolish the excessive secretion.

In considering the pathological anatomy, the physician must think in terms of both gross and microscopic changes in the gland for the two are interrelated. Furthermore, to obtain a proper understanding and evaluation of the various types of goiter one must constantly keep in mind the fundamental pathology, since a classification based on other than basic structural factors can be only confusing and misleading. Reference to the embryological development of the thyroid and a consideration of the evolution of the various types of goiter in the normal thyroid is not only

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helpful but essential to a clear understanding of the problem of hyperthyroidism.

The original thyroid anlage is composed of an undifferentiated, structureless mass of cells which (1) develop into small acini surrounded by a single layer of epithelial cells containing a characteristic colloid, and according to some authors (2) form nests of fetal epithelial cells—the interstitial cells of Woelfler or embryonal rests of Cohnheim—which remain scattered throughout the gland between the normal acini. The rests of Cohnheim do not take part in the formation of the normal thyroid acini or in the production of colloid but lie latent in most persons throughout life. Keeping in mind the two types of cells just mentioned, namely, (1) the acinar or parenchymal thyroid cell and (2) the interstitial or fetal cell, we may now consider the origin of the different kinds of goiter from these two types of epithelial cell.

TYPES OF HYPERPLASIA

Mild Hyperplasia of the Puberty Type is sometimes called the goiter of adolescence, or erroneously "physiological overactivity." It represents the first stage in the proliferation and hyperplasia of the normal acinar cell. The condition occurs usually in young girls at puberty or soon after. There is usually a mild hyperplasia and hypertrophy of the thyroid, with possibly a slight increase in the blood supply without, however, frank thrills and bruits. Microscopically, there is an increase in the size of the cells of the acini with a moderately increased alveolar infolding and occasionally an increase in the colloid content. The thyroid is fairly firm and elastic. Such a gland produces a mild hyperthyroidism which expresses itself in increased nervousness and irritability, a high-strung temperament, a mild emotionalism, a mild tachycardia, occasional palpitation, slight dyspnea, fatigability, vasomotor instability and a mild tremor. While the eyes may show increased brightness, there is usually absence of frank exophthalmos. The symptomatology tends to great variability and its relative mildness should be emphasized. In some cases it is almost entirely subjective and can be elicited only by painstaking and patient questioning. It is questionable whether one should regard the milder forms as due to true pathological changes or simply as expressions of transient, heightened, physiological activity. Many cases of this kind undergo spontaneous cure. However, it is by no means wise to assume that because of the comparatively mild

symptomatology and the slight enlargement of the thyroid that the patient will necessarily outgrow the condition.

Exophthalmic Goiter. (Synonyms: Von Basedow's disease, Graves' disease, diffuse hyperplasia, vascular goiter, etc.) This group represents the most complete expression of the advanced stages of hyperplasia and hyperthyroidism. The thyroid gland is symmetrically enlarged and presents a marked increase in its circulation as evidenced by the presence of thrills and bruits over the superior poles and often over the gland itself. The increased vascularity may even extend to the surrounding tissues. In the beginning, the gland is usually soft and elastic. Later it becomes firm and granular. The parenchymal or acinar cells enlarge, multiply and become tall and columnar. The acini lose their characteristic normal spherical configurations and assume various shapes with marked distortion and infolding of the alveolar walls. Furthermore, the colloid content of the acini is diminished. Groups of lymphocytes are occasionally present but are hardly characteristic.

Predisposing Causes. Sexual cycles—puberty and the menopause—undoubtedly play a part in some cases. In 1928, Warthin[1] described a type of person that by nature is predisposed to hyperthyroidism.

> The present day conception of Graves' disease is that of an extremely variable and broad syndrome. If we consider this clinical syndrome analytically we see that there is presented the clinical picture of a well-defined type of human individual—a distinct pathological constitution. This fact has not hitherto been recognized by American writers on goiter, but in Europe, both in Germany and in Italy, various writers are beginning to speak of the hyperthyroid constitution, (or Graves' constitution, as I have preferred to call it) in the absence of any positive proof that thyroid hypersecretion is its underlying etiological factor.
>
> The Graves' constitution individual presents a youthful build with a slender, delicate and soft skeleton, slender waist, . . . long slender fingers and toes with pointed terminal phalanges, abundant hair, and well-developed teeth and nails. The muscles are long and thin; there is underweight rather than overweight. . . . The facies is that of a bright-eyed, snappy, vivacious, quick-reactioned, ingenuous, often very attractive, youthful or childish appearance; . . . Muscle weakness is a common symptom. The skin is usually warm and moist, fine, delicate and translucent; . . . Hyperidrosis is common, and also dermatographism. . . . There is a marked instability of the central nervous system, in the form of increased sensibilities and emotional response, quickness of perception and reaction.

. . . In the majority of cases the marked irritability of the sympathetic system is a predominant characteristic. . . . Summing up the most striking and characteristic features of this constitution, we find them expressed in juvenile morphology and rapid functional reaction.

Racial factors, "autonomic imbalance," the hyperthyroid temperament and the so-called Graves' constitution as noted are important. Focal infection is sometimes mentioned as a cause though without compelling evidence. In the course of treatment, many patients have had various foci of infection removed: teeth have been needlessly sacrificed, sinuses drained, tonsils and adenoids and even gallbladders have been removed. In our opinion there is no evidence that focal sepsis predisposes to goiter. However, the role of sepsis and infection in exacerbating an existing hyperthyroidism is frequently borne out particularly during epidemics of influenza. The disease may affect several members of the same family. We have seen it in grandmother, mother and child. The sex element is attested by the great predominance of women over men.

Signs and Symptoms. Graves in his original description of exophthalmic goiter recognized a classical triad of cardinal symptoms—tachycardia, exophthalmos and goiter. Charcot added a fourth symptom—tremor.

Onset. The onset may be acute but more often it is insidious. When symptoms develop abruptly, they usually follow severe emotional strain such as fright, anger and profound grief particularly in persons conditioned and predisposed by "Graves' constitution." However, it is generally possible to uncover a latent period of mild hyperthyroidism antedating the sudden exacerbation. Remissions followed by recrudescences are common. Once the condition is frankly expressed, spontaneous and medical cures are rare and unless surgical relief is obtained, chronic invalidism with eventual physical dissolution or severe and even fatal crises may result. A classical, fully developed case presents little or no difficulty in diagnosis. However, in its incipiency, the condition is frequently overlooked. Hence one should watch for early signs and symptoms. It has been said that in its early stages, exophthalmic goiter is more a disease of the patient than of her organs. A patient's attitude, her approach to the physician, the dilated pupils, the restlessness, the purposeless rapid movements, the euphoria, the warm, moist, hands, the flushed face and the patient's haste may all be significant long before goiter can be detected. When to these suggestive signs are added unexplained loss of

weight, dispositional and temperamental change, emotional instability and uncontrolled energy, exophthalmic goiter may be lurking and should be suspected.

Early Vascular Signs and Symptoms. Increased pulse rate is frequently one of the earliest signs and may lead to "heart consciousness." (The average person at rest is of course ordinarily not conscious of his heart action.) In the beginning, the rate is regular and may rise from 80 to 90 and gradually increase to 120 or more. The heart may show little except an accelerated action. One should be careful in the evaluation of apparent murmurs since functional murmurs are fairly common. When true murmurs are present, they are due to other causes such as rheumatism and previous systemic infections. When the disease is fully expressed, visible cardiac pulsations over the precordium are greatly increased and the heart action is heaving and forceful. Likewise, the neck vessels throb forcibly. Thrills and bruits are commonly present over the thyroid particularly over the upper poles. They result from increased functional activity of the gland which, because of the hyperplasia, demands an increased blood supply.

Exophthalmos and Other Ocular Signs. The characteristic staring expression results from the protrusion of the eyeballs and the resultant exposure of the sclerae above and below the corneae (exophthalmos). In rare cases, severe conjunctivitis, corneal ulceration and panophthalmitis may develop. The principal characteristic eye signs in addition to the exophthalmos are von Graefe's sign, or the incoördination of the eyeball and eyelid when the patient glances downward; Stellwag's sign, or the widening of the palpebral fissure with infrequent winking; Moebius' sign, or the lack of power of convergence on near objects. The ocular signs result from weakness and incoördination of the extrinsic muscles of the eyes. The occurrence and unilateral exophthalmos while relatively uncommon is by no means rare. No satisfactory explanation exists for this peculiar phenomenon. (Fig. 1A and B.)

Goiter. The earliest cardiac and nervous symptoms usually antedate the appearance of goiter. In many cases thyroid enlargement may be mild or absent and varies widely in the same individual over a period of time. In the majority of cases, however, an enlarged gland can be demonstrated without difficulty and enlargements three or four times normal are not infrequent. It should be stressed that there is no quantitative relationship between the extent of thyroid

enlargement and the degree or duration of the symptomatology. Indeed palpable thyroid enlargement is not necessary to make a diagnosis.

A B

FIG. 1. S. H. A, age 28. Exophthalmic goiter with unilateral exophthalmos. B, age 29. Photo taken six months following bilateral resection. The right eye is still slightly prominent.

Tremor. The characteristic fine (though at times coarse) vertical tremor of the extended fingers is almost invariably present. It is involuntary, though accentuated by voluntary effort and averages seven to nine or more oscillations per second. Tremor may extend to the forearms, legs and in fact to practically all of the body musculature. A peculiar quivering of the lips when the patient speaks is frequently noticed in the unusually active case.

Other Signs and Symptoms. One of the earliest symptoms commonly encountered is weakness or asthenia. In fact it may take precedence over the customary nervousness. The patient complains of being tired most of the time and may not be refreshed by a night's rest. In severe cases, the muscular weakness is so disabling that the patient may feel as though her legs were unable to support her. Loss of weight is common especially when there is greatly increased metabolism. One should be doubly suspicious when the loss of weight occurs in the presence of a voracious appetite and good digestion. Flushing and sweating are common and pruritus is occasionally encountered. Acne, comedones and erythematous lesions of the skin are fairly common. Irritability of temper, dispositional change, depressions and moods are frequently present long before other more definite symptoms occur. Thus for example, a housewife without

apparent cause may become cross with her family or impatient with her children. Memory change is fairly common. The patient may forget her appointments and misplace ordinary objects of daily use.

A B

FIG. 2. J. P. A, age 44. Preoperative photo. Typical Graves' disease with classical symptoms. Basal metabolic rate +101 per cent. B, same patient, age 47. Photo taken three years following bilateral resection. Patient's health entirely restored.

Marked restlessness, disturbing dreams and great apprehension are other symptoms. The patient may dread the ordinary routine of life such as telephoning to and meeting her friends. She is very apt to look upon the serious side of life. Phobias are common such as the fear of going insane. Nausea, vomiting and severe diarrhea with great emaciation may occur in the highly toxic cases. These manifestations are to be regarded as ominous.

Course. The course varies. The onset is usually insidious. Remissions are fairly common but it is rare to have all of the symptoms disappear. The cases beginning in early adult life seem to progress less favorably than those developing in the later decades. If allowed to run its course, the disease is usually fatal. Death results from myocardial degeneration and cardiorespiratory failure. A number may end in "burnt-out" thyroids, indicating that the hyperthyroidism has spent itself. The patient, however, remains a permanent cardiovascular invalid. An occasional case may end in myxedema. (Fig. 2A and B.)

OTHER TYPES OF HYPERPLASIA

The task of detailing the signs and symptoms would be simple indeed if every case or even most of the cases that are seen in practice conformed to Graves' original description. We have described the milder forms of hyperplastic enlargement commonly referred to as puberty hyperplasia and the typical, full-blown hyperplasia known as Graves' disease (Basedow's disease or exophthalmic goiter). It naturally follows that there are certain intervening stages of differing severity in which the thyroid shows varying degrees of hyperplasia between the very mild and the extremely active and fulminating types of exophthalmic goiter. The clinical activity in these moderate hyperplasias varies within wide limits but in general it is proportional to the degree of the hyperplasia. Many of the symptoms of Graves' disease are present. However, they are present in less active and impressive form. Thus the pulse rate, loss of weight, tremor and general nervousness are not as severe. It is mainly by the diminished severity of the symptoms and the absence of the characteristic ocular signs that the moderate hyperplasias are differentiated from Graves' disease. Thrills and bruits, though they may be present, are likewise less marked than in Graves' disease.

ADENOMA OF THE THYROID (SYNONYM: NODULAR GOITER)

Thus far we have spoken only of the types of hyperthyroidism that result from hyperplasia of the alveolar cells and we recognized (1) the mild first stage or puberty hyperplasia, (2) the extreme hyperplasia of Graves' disease, and (3) the intervening types of moderate hyperplasia of varying degrees of clinical activity. Next we should consider a very different type of pathology, namely, the benign, new growths or adenomas which originate according to some authors not from the acinar cells but from the interacinar interstitial epithelial cell rests of Woelfler. These cells are present in all normal glands and because of their loose structure and the fact that they do not take part in the formation of normal thyroid acini, are regarded as lying dormant and inactive. They resemble embryonic thyroid cells and are, therefore, referred to as fetal cells. When one or more of these interstitial cell groups begin to grow, a mass of cells is developed. The mass which at first is microscopic in size, grows until it develops macroscopic proportions. Nodules may occur as single or multiple adenomas. As they increase in size, the

contiguous thyroid parenchymatous tissue is compressed and may undergo pressure atrophy. Gradually, through a deposit of connective tissue, a capsule forms about the newly developed tumor. Early encapsulation is characteristic of adenoma. In the beginning the adenoma is firm and smooth. However, since we are dealing with neoplasia, we may in time expect the usual sequences of neoplastic disease. First, fatty degeneration with central autolysis and fluid accumulation takes place (cyst-adenoma). This may in time be followed by hemorrhage into the interior (hemorrhagic cyst-adenoma), or the lesion may undergo fibrosis and calcification with marked thickening of the capsule. Finally, an appreciable number of adenomas of the thyroid, like adenomas elsewhere may undergo malignant degeneration (adenocarcinoma).

A different view of the etiology of thyroidal adenomas is that of Graham who presented a comprehensive paper on the relation of nodular goiter to neoplasia before the American Association for the Study of Goiter in 1929. In contrast to Woelfler's theory is that which ascribes the development of nodular goiters to a process of irregular hyperplasia and involution of the acinar cell. Graham believes that this idea is less speculative than the theory of origin from embryonal rests. The adult thyroidal epithelium involutes or reverts ("de-differentiates") to such a degree that it resembles the thyroid of a fetus. In brief, it assumes an embryonal character. Whatever the origin, whether from Woelfler's embryonal rests or by a process of "de-differentiation" (Graham)[2] of the acinar cell, basic changes must occur in the original tissue before neoplasia results.

The adenomas per se are responsible for the hyperthyroidism. They secrete a toxin which, if not actually chemically pure thyroxin, bears a close relationship to it. As the adenomas grow and develop, they elaborate increasing amounts of secretion and cause varying degrees of hyperthyroidism, mild to moderate or severe. The reason for the development of an adenoma from the dormant interstitial cells is not apparent. However, certain predisposing or exciting causes are recognized. First in importance is pregnancy which unquestionably bears a very close relationship to their development and which we shall consider in detail later under the heading, "Hyperthyroidism and Pregnancy."

During puberty and the menopause, certain endocrine disturbances involving the ovarian, thyroid and adrenal functions undoubtedly play a part in precipitating the appearance of adenomas.

Adenoma rarely occurs before puberty but is not uncommon in the postpuberty period. It is frequently seen during the menopause. Focal infection has been suggested as a factor in the etiology. It is

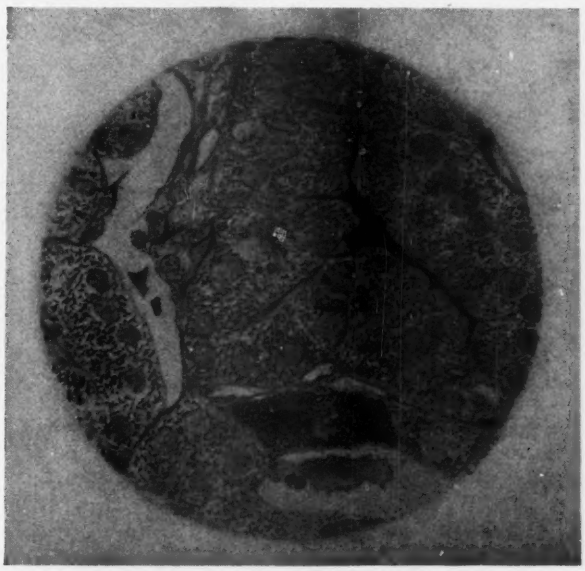


FIG. 3. Section of active hyperplasia—exophthalmic goiter—showing great numbers of mitochondria.

difficult to understand how an infection—focal or otherwise—could have any influence in the production of neoplastic disease. Furthermore, eradication of the foci is without benefit on the progress of the lesions. On the contrary, operative measures, such as tonsillectomy or the draining of sinus infections, invariably exacerbate the hyperthyroidism.

The number of adenomas that may be present in a single thyroid varies greatly from a solitary tumor to several hundred nodules. Likewise there is a great variability in the symptoms dependent upon the structure and cellular activity of the tumors. The recent young, more active nodules produce the most striking symptoms while the colloid and degenerative types produce less definite and active symptoms.

Since adenomas at their inception are microscopic in size, they may grow and enlarge considerably before they are suspected or discovered. Obviously then, a patient may show symptoms of hyperthyroidism long before palpable nodules are present. By careful staining methods (Goetsch)[3] it has been shown that toxic adenomas contain an abundance of structures called mitochondria which are

granular or rod-like in appearance. They have a great affinity for acid fuchsin which gives them a brilliant red color, and they are indices of protoplasmic activity. Accordingly large numbers of

FIG. 4. F. M., age 23. Discrete nodule (adenoma) in right lobe. Mild hyperthyroidism.

mitochondria are found in the thyroid cell in the severer grades of hyperthyroidism. (Fig. 3.)

Signs and Symptoms. In early cases, the patient may appear to be somewhat thin and sallow and possibly mildly underweight. The hands and feet are usually cold and clammy and there is ordinarily a tremor of the extended fingers. As the disease progresses, chronic fatigue and weakness become prominent symptoms. The spirit is willing but the strength is lacking. Emotionalism, crying spells without real provocation, hypersensitivity, depressions, moods and even melancholia may supervene. Lack of concentration and forgetfulness are quite common. Other symptoms are dispositional change such as irritability and flights of temper. Many patients complain of inward nervousness, a bizarre feeling of heaviness or uneasiness about the heart among other peculiar neurotic "inward" symptoms. Lability of the pulse rate is a rather characteristic finding. Sooner or later changes in the thyroid can be demonstrated. Careful palpation may

at first reveal the presence of a tiny nodule in the thyroid long before any visible enlargement is noticed. The small, early nodule is usually tender to palpation. This characteristic tenderness is of especial significance in demonstrating the incipient nodule particularly in the borderline or obscure case. As the nodule enlarges and especially when additional nodules are discovered, the hyperthyroidism is increased. The gland enlarges and unlike hyperplastic goiter loses its normal symmetry. (Fig. 4.)

CARDIAC SYMPTOMS—THE ROLE OF THE THYROID HORMONE

Continuous hyperthyroidism whether due to hyperplasia or adenoma leads to progressive myocardial insufficiency. In exophthalmic goiter, the toxemia is usually more severe and serious cardiac damage with incapacitation results earlier than in adenoma in which toxemia is usually less marked but more prolonged. However, eventually the cardiac damage and incapacitation may be quite as severe. This may in part be due to the average greater age of patients with adenoma.

Following the initial tachycardia, the ensuing cardiac signs and symptoms of dyspnea, irregularities in the pulse rate and extra systoles are followed by auricular fibrillation and occasionally by flutter. As the hyperthyroidism continues, the preceding symptoms are accentuated and a train of events leading to so-called "goiter heart" develops. The first indication of threatened cardiac incompetency is the presence of a few scattered and inconstant basal rales. Signs of incompetency such as an early ankle and shin edema gradually appear. At first the edema vanishes after a night's rest. Gradually the edema progresses upward along the legs and thighs. Abdominal ascites, pleural and pericardial effusion, cardiac dilatation and hypertrophy supervene and may terminate in general anasarca and death from cardiac failure.

During the progress of the preceding symptoms, significant rises in the blood pressure take place. In the early stages, the blood pressure rises only slightly if at all and a wide pulse pressure is maintained. As time elapses the blood pressure gradually climbs, arteriosclerotic changes in the blood vessels appear and unless the hyperthyroidism is relieved by operation, a permanent hypertension with a lowered pulse pressure may result and remain permanently whether operation is performed or not.

A considerable group of thyroid patients is one in which the hyperthyroidism is masked and in which a provisional diagnosis of

organic cardiac disease is made. We refer to a group of middle-aged patients who are obviously not suffering from Graves' disease but who have been suffering over long periods of time, possibly years, from chronic, mild, unrecognized hyperthyroidism due to adenomas that escaped detection and in whom the hyperthyroidism is overshadowed by the predominant cardiac symptoms. Too often they are treated as primary cardiacs and little if any attention is paid to the thyroid. Digitalis, rest and supportive treatment fail to correct the condition. Unless a careful history revealing thyroid symptoms and signs and the discovery on careful repeated search of one or more nodules in the thyroid indicate the true condition, such diagnostic aids as the electrocardiogram and the basal metabolic rate are of questionable aid. Patients suffering from primary, organic, cardiac disease besides showing electrocardiographic evidences of myocardial degeneration may, like the hyperthyroid patient, also have elevated basal metabolism. It follows that in every elderly patient showing tachycardia, cardiac irregularity or evidences of failing compensation of obscure origin, we should be on constant watch for evidences of hyperthyroidism. In exceptional cases the *x*-ray may reveal the presence of a substernal goiter.

The importance of the recognition of hyperthyroidism superimposed on a heart that is already damaged by rheumatic disease and hypertension can hardly be overemphasized. The hyperthyroidism accompanying such conditions may be the prime factor in causing the heart to become incompetent. Early operation may once again render the heart competent.

Although the association of proven coronary disease with hyperthyroidism is in our experience rather rare, anginal attacks are by no means infrequent. Occasionally, a patient may give a story of severe cardiac or substernal pain which radiates to the shoulder and even into the forearm. However, electrocardiographic tracings fail to establish coronary disease and operation is followed by disappearance of the symptoms. Various hypotheses have been advanced to explain cardiac pain and anginal attacks in the thyrocardiac. None of them is entirely satisfactory. It has been suggested that dilatation and hypertrophy of the heart leads to ischemia of the heart musculature from stretching of the nutrient coronary vessels. Pain is said to be the result of the ischemia.

It has long been appreciated both clinically and experimentally that the heart action is peculiarly sensitive and responsive to the toxemia of hyperthyroidism. The exact mechanism of the production

of cardiac damage is not entirely clear. It is widely held that the myocardium is peculiarly sensitive and vulnerable to the thyroid hormone and that the cardiac manifestations result from the specific, destructive action of the thyroid toxin on the myocardium per se. However, in patients dying in the terminal phases of hyperthyroidism or from crisis, the heart muscle does not show any definite structural changes referable to a specific toxin. There are no characteristic basic findings that differentiate the heart of the patient succumbing to hyperthyroidism from one dying of cardiac failure from other commonly recognized causes.

Another view attributes the cardiac damage to accelerated circulation caused by increased metabolism and the need for increased amounts of oxygen. The heart is overworked and eventually becomes exhausted in its struggle against the continuous overloading in consequence of the elevated metabolism. In this connection, it may be mentioned that an appreciable number of unrecognized and obscure cases of goiter present cardiac arythmias and other evidences of cardiac damage as the first prominent symptoms. Over long periods of time, even years, there may have been absence of heart consciousness. The pulse and metabolic rates may have remained within normal limits, the weight may not have varied greatly and the characteristic symptoms of hyperthyroidism may have been minimal and thus escaped notice. It would seem logical to attribute the myocardial effects in these cases to a specific destructive action of the long continued, though mild, toxemia rather than to overwork. The rapid improvement in cardiac function that almost invariably follows successful thyroidectomy would seem to fortify this belief. Hearts damaged from causes other than hyperthyroidism are prone to remain damaged. The lowering of the glycogen content of the myocardium in hyperthyroidism is said to further predispose the myocardium to damage. Moreover, some observers attribute thr tachycardia and other cardiac manifestations to stimulation of the sympathetic nervous system by the thyroid hormone. It is very probable that the cause of the myocardial damage is to be sought not in one but rather in a combination of several factors. Whatever the mechanism, the surgeon's objective is to eliminate the thyrotoxicosis by adequate thyroid resection. This remains the best, in fact the only means of restoring cardiac function.

From the foregoing, it is clear that hyperthyroidism regardless of its etiology produces a large number of characteristic general

signs and symptoms. Some authors regard hyperplastic goiter and toxic adenoma as identical. We feel, however, that in matters of origin, symptomatology and fundamental pathology there are a sufficient number of differential points to classify as separate entities. These are contrasted in the following outline:

EXOPHTHALMIC GOITER	ADENOMA
The disease develops rapidly and continues at a quick tempo.	The symptoms develop insidiously and advance more slowly.
It is not uncommon under twenty years of age.	It is relatively uncommon before twenty years of age.
There is a diffuse, uniform enlargement of the thyroid. Normal symmetry is preserved. The gland is smooth, rubbery and resilient in consistency.	There is greater variation in the size of the gland. Single or multiple nodules of varying size and consistency are present and the normal symmetry is consequently lost.
There is generally rapid development of the symptoms. Tremor is marked and may extend to the forearm and even to the entire body. The nervous symptoms particularly the objective ones are pronounced. Restlessness is severe. Mental manifestations bordering on psychosis are not infrequent.	Symptoms develop more slowly. Tremor though present is less pronounced. Symptoms are milder and not as easily elicited. Restlessness is less obvious. Mental symptoms, while fairly frequent, are longer delayed.
The appetite is greatly increased. Loss of weight may be rapid and extreme. Vomiting and diarrhea are frequent. Acidosis and crises occur oftener.	The appetite is more variable. The loss of weight is usually less rapid and extensive. Vomiting and diarrhea are less frequent. Acidosis and crises are not as common.
Vasomotor instability, increased perspiration, flushing and heat intolerance are common.	These symptoms are less pronounced. Heat intolerance is less marked.
Pressure symptoms and mechanical factors are absent. The voice is not impaired. The goiter does not invade the mediastinum.	Pressure symptoms, deviation and constriction of the trachea are frequent. The voice may become husky from pressure on the recurrent nerve. A dry cough is common. Mediastinal encroachment is not unusual.
The pulse is generally higher—commonly 120 to 140. The pulse pressure is moderately increased.	The pulse is generally lower—commonly 90 to 110. The pulse pressure is greatly increased. Arhy-

EXOPHTHALMIC GOITER	ADENOMA
Arhythmias and fibrillation are fairly frequent.	thmias and fibrillation are more frequent.
A characteristic "drive" may be present.	Chronic fatigue is more frequent.
Ocular signs are characteristic. Thrills and bruits are commonly present.	Ocular and vascular signs are uniformly and characteristically absent.
The basal metabolic rates are generally higher.	The basal metabolic rates are more variable and tend to be lower.
The patient is more rapidly incapacitated.	Incapacitation takes place less rapidly.
Malignancy is rarely if ever encountered.	Malignant degeneration occurs in 3 to 4 per cent of cases.

DIFFERENTIAL DIAGNOSIS

While frank hyperthyroidism is usually recognized easily, there are certain conditions which may be confused with early hyperthyroidism.

Tuberculosis. Among the most important is tuberculosis. The familiar findings of fatigue, asthenia, lability of pulse rate, weakness, mild nervousness and vasomotor instability with increased perspiration might lead one to confuse early hyperthyroidism with incipient tuberculosis. The findings in the thyroid may not be convincing and a careful history, physical examination and *x*-ray studies may fail to differentiate the two. In a fair number of cases, the hyperthyroid patient may be detected from one suffering from incipient pulmonary tuberculosis by her constitutional response to adrenalin. (Goetsch).[4]

Sympathicotonia. The so-called sympathicotonic type of functional neurosis presents many signs and symptoms which overlap into the field of true hyperthyroidism. The syndrome is characterized by a highly sensitive nervous system. The common findings are a highly nervous state, asthenia, vasomotor instability, tachycardia, increased perspiration, tremor and mild emotionalism. The tachycardia is especially marked when the patient is under emotional strain and there is consequently great lability of the pulse rate. Frequently, phobias such as fears of fainting, of collapsing in the street or inability to take care of oneself unless accompanied by another person are superimposed. The symptoms may simulate

an anxiety neurosis and are generally of bad prognostic significance and very difficult to treat and eliminate.

The sympathicotonic individual can usually be differentiated from the true hyperthyroid by the fact that the nervousness is familial and has been present as a rule as long as the patient can remember, whereas in hyperthyroidism the complaint usually but not invariably has a definite beginning. Furthermore the complaint of the neurasthenic and sympathicotonic patient is rambling and bizarre; it keeps changing and becomes more and more involved. The true hyperthyroid patient on the contrary is noted for the consistency of his story.

The differential findings in the thyroid gland assume compelling significance in culling the neuroses from the hyperthyroid states. The gland is rarely enlarged in the neurasthenic and there are no findings such as nodules or increased circulation, whereas it is the exception to find real and definite hyperthyroidism unassociated with either some glandular enlargement, the presence of one or more nodules or evidences of increased vascularity. The basal metabolic rate in the neuroses is commonly normal or even below normal, whereas in hyperthyroidism the rate is usually but not necessarily elevated in proportion to the degree of the hyperthyroidism.

Neurocirculatory Asthenia—Pseudo-Graves' Syndrome. Patients suffering from so-called neurocirculatory asthenia present difficulties in the differential diagnosis. These individuals are sympathicotonic by nature. As children, they are delicate. They lack physical strength and hence do not choose strenuous occupations. They lack ambition and generally feel inferior. The common symptoms are palpitation, increased perspiration and extreme fatigue. Tremor and dermatographism are characteristically present. Very occasionally a sympathicotonic patient may show, in addition to the previously enumerated symptoms, some brightness and even an increased prominence of the eyes, and the thyroid though not enlarged may present minor but definite thrills and bruits such as one finds in Graves' disease. Briefly, the patient looks like one suffering from an early exophthalmic goiter. A number of terms have been applied by various authors—autonomic imbalance, pseudo-Graves' syndrome, effort syndrome and irritable heart. The important characteristics are the absence of an enlarged thyroid and the consistently normal metabolism. The condition is difficult to remedy and surgical attack on the thyroid should be scrupulously avoided.

It is apparent that the separation of the neuroses from true hyperthyroidism cannot be done quickly and with unfailing accuracy. The diagnostic aids are limited and give us little real help in differentiating the truly hyperthyroid patient for whom surgery is indicated and curative from the functional neurotic for whom surgery is contraindicated and harmful. It should be stressed that when a border-line case is suspected of hyperthyroidism, nothing can be lost by a period of waiting and observation at intervals. Since hyperthyroidism is a progressive disease, time will reveal the true character of the complaint and permit the development of objective diagnostic findings in the thyroid. Useless operations will be avoided. A therapeutic test will occasionally be helpful especially in dealing with suspected hyperplasia. Lugol's solution may be given in routine fashion for a week. If the patient is a neurotic, no harm will be done; and if she is suffering from hyperthyroidism, a favorable response may indicate the advisability of operative treatment.

PRESSURE EFFECTS OF GOITER

In the matter of mechanical effects, we need consider practically only the adenomatous goiter. Hyperplastic goiter—whether a mild hyperplasia or exophthalmic goiter—rarely causes any serious mechanical embarrassment. The pathology involves the gland as a whole rather than any particular part. Consequently, the enlargement extends in all directions, symmetry is maintained and the goiter even when large does not extend downwards into the mediastinum. Hence, serious mechanical difficulties involving tracheal displacement or narrowing rarely if ever occur, and respiratory embarrassment such as suffocation and stridor are practically never encountered. Very occasionally a moderately large hyperplastic goiter, particularly if it is present in a short stout neck, may be engaging the thoracic inlet too snugly causing some distention of the superficial veins of the neck leading to a sense of fulness and pressure in the head or an occasional headache. In general, we need not worry about serious mechanical difficulties provided we realize that we are dealing with a hyperplastic goiter.

With adenoma, however, the problem is entirely different. A large adenomatous goiter even though it maintains a cervical position may lead to troublesome and serious mechanical difficulties. Thus a large tumor of either the left or the right lobe may so displace

the trachea to one or the other side that the transverse diameter is greatly narrowed at the point of maximum pressure. The patient complains of increasing difficulties in breathing and if a respiratory

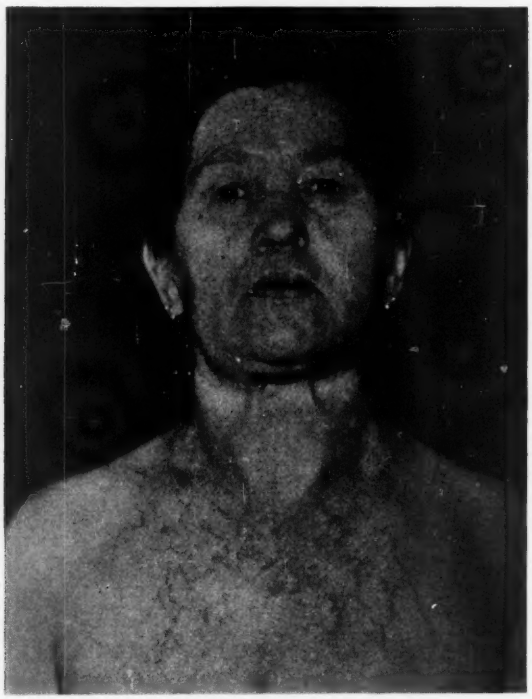
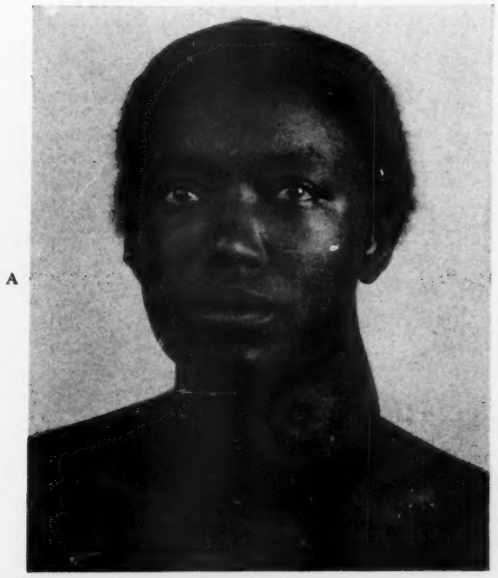


FIG. 5. C. E., age 53. Long standing adenomatous goiter with marked pressure effects and typical caput medusae.

infection should supervene, severe dyspnea may result. In fact, the congested tracheal mucosa incidental to respiratory infection may so compromise the already diminished lumen that acute suffocation requiring emergency measures may supervene. The preceding condition may be further aggravated if a large adenomatous mass on one side opposes a small, firm nodule on the other side. This is particularly true if the smaller mass is firm and hard enough to cause pressure softening of one or more tracheal rings.

The most frequent cause of serious pressure symptoms is the goiter that has become partly or entirely intrathoracic. The most common is that due to an adenomatous nodule at one or the other of the lower poles or in the isthmus. Gradually through gravity and enlargement it may descend beneath the clavicle or sternum into the mediastinum. To begin with, the nodule is extrathoracic in position. It may in time be alternatingly aspirated into and expelled

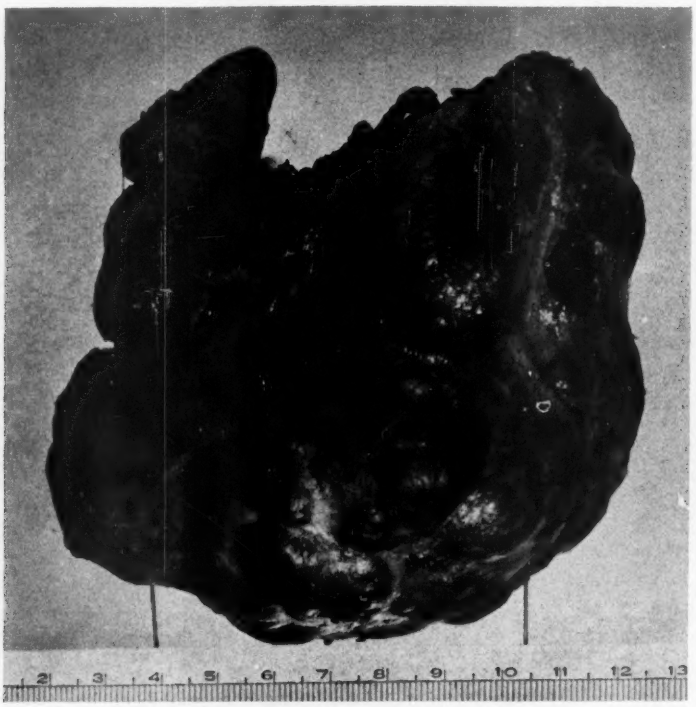


A

B

FIG. 6. M. B., age 42. A, huge adenomatous goiter with tracheal displacement and pressure effects. B, *x*-ray film showing marked displacement of the trachea. C, excised tumor of left lobe.

from the mediastinum by deep inspirations and pressure of the cervical musculature resulting in the condition called wandering goiter. Serious difficulties may not arise until it attains such a size



C

FIG. 6. For descriptive legend see opposite page.

that expulsion can no longer take place. Gradually increasing dyspnea or suffocation may supervene since the more the goiter enlarges the more it is restricted by the unyielding thorax. If perchance the intrathoracic goiter should be bilateral, pressure is exerted in two directions and a scabbard trachea results. In addition to direct mechanical effects on the trachea per se, prolonged pressure on one or the other recurrent nerves may result in abductor paresis producing hoarseness and adding further to the respiratory distress. Finally, pressure on the jugular veins may so interfere with the venous return that the superficial veins are rendered prominent from engorgement, producing eventually the caput medusae so typical of large retrosternal intrathoracic goiter. This venous phenomenon represents a compensatory mechanism on the part of the veins of the neck and upper thoracic region. (Fig. 5.)

For the most part, an intrathoracic goiter is only partly so. More often the tumor is both cervical and retrosternal or subcla-

vicular in position and the upper limits can be palpated particularly when the patient swallows or is made to cough forcibly. In the comparatively rare condition in which the goiter is completely intrathoracic, no portion rises into the cervical region under any conditions and its pedicle or connection with the thyroid proper may easily escape detection.

Whenever on the basis of the foregoing signs and symptoms, substernal or intrathoracic goiter is suspected, one should look for evidences of hyperthyroidism in the history and physical examination. Percussion may reveal more or less retrosternal dullness over the mass. Finally, the *x*-ray is of invaluable help. An anteroposterior view should be taken. This will reveal any lateral displacement or constriction and shape of the trachea, whether S-shaped, spiral or otherwise. Since a substernal goiter arising from the isthmus may retain a median position, a lateral *x*-ray view will usually disclose whether there is narrowing or compression of the trachea in the anteroposterior diameter or whether the goiter has insinuated itself between the trachea and the esophagus.

Occasionally, particularly with a relatively non-toxic, intrathoracic, adenomatous goiter that has produced little if any hyperthyroidism, the differential diagnosis of goiter and mediastinal neoplasm such as teratoma or thymoma becomes difficult. However, an adenoma tends to maintain its smooth discrete outline. Other tumors are usually less discrete. Fluoroscopy is of invaluable aid. It may establish the diagnosis in doubtful cases since practically all intrathoracic goiters maintain connection with the thyroid and hence tend to move upward on swallowing as distinct from most other mediastinal tumors which tend to remain fixed. (Fig. 6A, B and C.)

TESTS FOR HYPERTHYROIDISM

A few clinical laboratory findings are helpful in diagnosis. In active hyperthyroidism there is an actual and a relative increase in the mononuclear leucocytes. They may reach 60 to 70 per cent or the total count. With this increase in the mononuclear count, there is a corresponding decrease in the polymorphonuclear cells to 35 of 40 per cent of the total. These findings were first pointed out by Kocher who was led to attach considerable prognostic value to the degree of lymphocytosis. The higher counts indicated to him the less favorable cases of hyperthyroidism.

Blood Sugar Level. A relative diminution in the sugar content of the blood is commonly observed in hyperactive cases and varies with the degree of the hyperthyroidism. In severe cases the sugar level may fall to 100 or even 80 mg. of sugar per 100 c.c. of blood.

Blood Cholesterol. In general the blood cholesterol varies inversely with the extent of the hyperthyroidism and directly with the degree of the hypothyroidism. However, the inverse relationship is not constant and the cholesterol determination, therefore, has been only of passing significance as a diagnostic aid.

Urinary Findings. There are no characteristic changes in the urine until late degenerative effects in the cardiovascular-renal system are produced. In some extremely active cases, glycosuria may occur as a result of the rapid mobilization of glucose in the body with resultant overflow in the urine. It occurs oftenest in the severe hyperplasias and usually disappears after operation. It must be differentiated from true diabetes, in which the administration of glucose is followed by a rapid rise and a relatively slow fall in the blood sugar level, whereas in hyperthyroidism the blood sugar under similar conditions also rises but is less sustained than in diabetes.

Basal Metabolism. In addition to information gained from the history, symptoms and physical signs, certain tests are helpful in the detection of hyperthyroidism and hypothyroidism. Friedrich von Müller and Magnus-Levy were the first to point out that an increase in general metabolism is one of the most significant features of hyperthyroidism and that a decrease in the metabolism is characteristic of hypothyroidism. von Müller discovered that his patients with exophthalmic goiter not only consumed more food than was required to meet their caloric needs, but that they were excreting more nitrogen than they were receiving in their food. The patients were actually in a condition of relative inanition notwithstanding that they were consuming far more food than normal persons of the same age and weight. These facts were interpreted as due to a regulatory action of the thyroid hormone on the fundamental metabolic processes.

Very soon after these discoveries, attempts were made to measure the heat production and basal metabolism in patients under various conditions of health and disease. By the term "basal metabolic rate" of an organism we mean the minimal heat production of that organism measured from 12 to 18 hours after the inges-

tion of food under complete rest. It is not within the scope of this paper to go into the various details of determining the basal metabolic rate since devices for its estimation have been in use for many years in all well-conducted hospitals. We shall rather content ourselves with a consideration of its application to clinical practice.

The basal metabolic rate when accurately determined is of great value in the diagnosis and treatment of thyroid disorders.[5] However, it is not an infallible index and should never be the only guide in estimating the degree of overactivity. Zuntz was the first to realize the importance of absolute muscular repose in determining the true basal metabolism and Magnus-Levy showed the influence of various factors such as age, sex, weight, excitement, menstruation and pregnancy. In general it is accepted that the level of the basal metabolism is a valuable guide in judging the degree of the hyperthyroidism. This supposition must, however, not be accepted without reserve especially when only a single rate has been determined. First of all, there is always the danger of error through leakage and faulty technic. Also one must be on the alert for conditions such as fevers, tuberculosis, diabetes, various anemias and pituitary disorders which tend to increase metabolism.

In cases of adenoma the basal metabolic rate is helpful in estimating the degree of the toxemia and in general it parallels the clinical activity. However, we should bear in mind that chronic toxemia in long standing cases may have damaged the nervous system, heart and kidneys to such an extent that the metabolic activity may be depressed to a relatively normal or even subnormal level. A favorable rate might, therefore, be a poor criterion of the patient's ability to withstand operation. A careful evaluation of the history and the physical status of the patient are of relatively greater importance.

In numerous cases of adenoma, the symptoms are for the most part subjective in character and difficult to elicit. There may be absence of tremor and objective nervousness and the basal metabolic rates may well be within normal limits or actually below normal. Excepting for the presence of one or more nodules in the thyroid, one would be prone to dismiss such patients as free from hyperthyroidism. Provided there are no clinical evidences of hypothyroidism, we have not been deterred from giving such patients the benefits of operation and have invariably noticed that operation is followed by

a prompt cessation of the subjective nervousness and a general feeling of well-being. Incidentally following operation, depressed metabolic rates tend to rise due in all likelihood to a rehabilitation of the normal thyroid elements after pressure and possibly other inhibiting effects of the adenoma have been removed. A basal metabolic rate may thus indicate that the oxidative processes may be normal without, however, indicating other altered functions or dysthyroidism that can be relieved by operation. This brings to mind the terms "toxic" and "non-toxic" adenoma. It is abundantly clear from the literature and discussions among physicians that these terms mean different things to different people. To some the word "toxic" is associated with increased metabolism only, while to others it signifies the presence of symptoms. It is obvious that a great deal of confusion may result and that certain patients with toxic adenomas as judged from the symptoms and signs might be denied the benefits of operative treatment, if one were to wait for increased metabolism to establish the diagnosis.

It is in the cases of hyperthyroidism due to the higher grades of hyperplasia as found in exophthalmic goiter that the basal metabolic rate is especially helpful. The basal metabolic rate in exophthalmic goiter as with active adenoma commonly parallels the degree of the hyperthyroidism. In general, but particularly in recent and acute cases, the higher the rate, the greater the hyperthyroidism. However, one cannot be arbitrary in setting a definite level beyond which the basal metabolic rate indicates a danger of severe postoperative hyperthyroidism. Nevertheless, when the history and the clinical status of the patient indicate a severe thyroid toxemia, a high basal metabolic rate in excess of +50 or +60 per cent should tend to make the surgeon cautious, lest by too extensive an operation he bring about a critical reaction or possible fatality. Preliminary ligations or stage operations may become necessary.

In chronic hyperthyroidism with marked emaciation, one should realize the influence of poor nutrition in depressing heat production. While the basal metabolic rate may give us an index of the amount of combustion that is taking place, it does not give us a clue to the amount of degeneration that may have taken place in the cardiovascular system, the kidneys, nervous system and other organs which so largely determine the patient's ability to withstand operative procedures. Therefore, in cases of long standing hyperthyroidism with severe cardiac damage, a favorable metabolic rate may mis-

lead the surgeon into attempting too extensive an operation and courting an unexpected fatality.

It is obvious that the basal metabolic rate can in no manner replace the necessity for detailed clinical study of the individual case, that a normal rate does not exclude hyperthyroidism and that an elevated rate by no means always indicates hyperthyroidism. Due regard must always be had for the clinical picture and when this necessity is more generally appreciated, physicians will be less prone to demand routine basal metabolic rate determinations as the all-important considerations.

ACIDOSIS—THE ROLE OF CARBOHYDRATE METABOLISM

It is a rather frequent experience among thyroid surgeons that a more or less serious acidosis is prone to occur in the early postoperative period especially when dealing with the higher grades of hyperthyroidism. The acidosis manifests itself clinically by a number of annoying symptoms such as extreme nervousness and restlessnesss, nausea and vomiting, headache, diarrhea, dry, parched tongue, acetone odor to the breath and acetone bodies in the urine. When these symptoms and findings are superimposed upon those due more directly to the hyperthyroidism such as severe tachycardia and a rising temperature, the surgeon is confronted with a syndrome fraught with great anxiety and very distressing to the patient.

The relationship of the thyroid to carbohydrate metabolism offers a probable clue to the etiology of the disturbing acidosis, since of the ductless glands the thyroid and adrenals are probably the most directly involved in disturbances of carbohydrate metabolism. Thus, for example Kuriyama discovered that the feeding of thyroid extract to white rats produced a diminished glycogen content of the liver. There are other facts indicating that excessive thyroid secretion and adrenalin mobilize glycogen and produce thereby an increased sugar content in the blood provided, of course, that there is a sufficient glycogen reserve. Moreover, prolonged hyperthyroidism causes a marked loss of body weight and an accompanying lowering of the glycogen reserve. It is our experience that acidosis is prone to occur particularly after operation as a result of this depletion.

Previous to 1922 in our search for an explanation for the postoperative reactions, some interesting and we believe enlightening investigations were made at our Clinic (Goetsch and Browder)[6] on

the sugar content of the blood of patients with varying degrees of hyperthyroidism as contrasted with those of normal controls. Since the thyroid is so intimately bound up with metabolism, we should expect varying degrees of disturbances in the metabolism proportionate to the amount of thyroid hormone in the circulation. First we studied the behavior of the blood sugar before, during and following operation. It was demonstrated that there is an early, rather definite rise of blood sugar as a normal response to the factors encountered in the operation such as anesthesia, trauma, and emotional disturbances whether in the hyperthyroid or the normal patient. The only significant difference noticed was the fact that in the normal control individual, the blood sugar rises gradually and over a longer period and only gradually falls to the normal level in two to three or more days, whereas in the hyperthyroid individual, the initial rise in blood sugar is more abrupt, is not so high and then falls promptly to the preoperative level or lower, often in one-half hour or less. It was found that in the more severe grades of hyperthyroidism, the hyperglycemic curve does not reach as high a level and falls even more rapidly, suggesting that in the very active cases there is a correspondingly greater depletion of the glycogen reserve and a diminished response to the factors calling forth glucose.

An interesting relationship between the degrees of hyperglycemia and the acetonuria and acidosis seemed to be present. Thus in those cases of marked hyperthyroidism in which the blood sugar curve was low and tended to fall promptly to normal or lower, the acidosis was correspondingly more severe. This suggested that when the glycogen reserve is low, the defense factors against acidosis are lacking and lead to the development of clinical symptoms and acetonuria. Conversely, when the blood sugar curve remained high and sustained following operation, only slight or no evidences of acetone or acidosis occurred. Therefore, there may well be a relationship between the sugar content of the blood and the appearance of acidosis. Indeed, our studies seemed to indicate that the higher the degree of hyperthyroidism, the less the hyperglycemia and the greater the likelihood of acidosis following operation. It seems probable, too, that when there is a liberal carbohydrate reserve in the form of glycogen, fats and proteins are spared while the glycogen is consumed and acidosis consequently minimized or prevented.

The foregoing considerations confirm on physiological grounds what has long been appreciated clinically, namely, that carbo-

hydrates play a major role in the physiology of the hyperthyroid patient. In preparation for operation, a liberal carbohydrate diet is very helpful in fortifying the glycogen reserve. Moreover, intravenous glucose injections are almost specific in the control of acidosis before and following operations on the thyroid.

Crisis. While it may be said that the physiology of the thyroid is as well understood as that of any endocrine, there is much regarding the pathological physiology of the thyroid that still remains in the realm of the speculative. This is particularly true of a curious and fulminating phenomenon known as thyroid crisis or storm, which occasionally develops spontaneously in the course of severe hyperthyroidism and which is still a major factor in deaths from hyperthyroidism. Fortunately in the numerous cases of hyperthyroidism that are observed and treated in a busy thyroid clinic, this ominous development is relatively infrequent. Unfortunately, however, the signals indicating impending storm are few and not clearly drawn. Certain facts and circumstances are, however, indicative. Predisposing factors to crisis in the course of a progressive hyperthyroidism are marked accentuation of mental states such as the emotions of fear, anger, worry and extreme anxiety. When these are coupled with a constantly and rapidly increasing rise in the metabolic and pulse rates, marked loss of weight, various phobias, mental deterioration and particularly nausea and vomiting, one should be especially vigilant with regard to a lurking crisis.

When crisis is impending, one notices a sudden accentuation of the physical, mental and nervous states of the patient. The general restlessness, the pulse rate and the temperature increase rapidly. In short the picture presented is one of an alarming exaggeration of all of the patient's previous signs of hyperthyroidism. His appearance indicates a condition of profound toxemia. Something seems to be eating at his very vitals and one gathers that a poison of unusual virulence is destroying him. Not infrequently, evidences of acidosis with acetone in the urine, acetone breath, severe vomiting and diarrhea add to the gravity of the picture. However, urinary findings may be singularly absent. As the objective findings—pulse, temperature and respirations—increase with alarming rapidity, the mental alertness and physical restlessness are maintained and may even be heightened until such time as an ominous mental dullness and general inertia and apathy indicate an impending dissolution. Unless the rapid march of events is halted, the patient lapses into delirium,

semi-consciousness, and cyanosis and dies in a state of hyperpyrexia (105 to 108°F.) and coma with a corresponding increase in both the pulse and respiratory rates.

Thyroid storm or crisis as described may also follow operations on the thyroid with similar critical developments. Postoperatively as preoperatively the warning signals may be indefinite and inconstant but just as we recognize certain predisposing causes in the spontaneous development of crisis or thyroid storm, we recognize certain dangers in the patient who has been submitted to surgical treatment. Extreme toxicity, unusually high metabolic rates, marked emaciation and mental deterioration are predisposing factors. The incorrect use of iodine in the medical treatment, particularly if given over prolonged periods, seems to predispose in a large percentage of cases. Moreover, patients who in spite of the standardized preliminary treatment with iodine fail to improve noticeably and who, following the preoperative iodinization maintain a high degree of hyperthyroidism with little if any drop in the metabolic and pulse rates, seem particularly liable to develop crisis. Then again the basal metabolic rate is no real criterion since crisis may occasionally follow operations on patients with relatively low metabolic rates as for example in those with long-standing, chronic adenomatous goiters. While stage operations undoubtedly tend to diminish the incidence of crisis, the type and extent of operation by no means always determines the likelihood of the development or absence of crisis. Deaths from crisis may occur after minimal procedures such as the ligation of a single, superior thyroid artery. On the other hand so-called bad-risk patients frequently go through extensive operations without untoward postoperative reactions.

The interrelationships of the sympathetic nervous system, adrenals and the thyroid have long been emphasized by Crile and studies have been made in our clinic which showed that the characteristic postoperative reactions such as restlessness, hyperpyrexia, tachycardia and other features of a severe reaction can be reproduced by the subcutaneous injections of adrenalin (Goetsch & Ritzmann).[7] Moreover pulmonary edema and tracheitis similar to that encountered in crisis may be caused in experimental animals by injection of adrenalin. It seems entirely probable, therefore, that crisis may in part at least be an expression of adrenal hyperactivity.

In a certain number of cases of postoperative crisis, jaundice and other manifestations of disturbed hepatic function have been noted

clinically and varying amounts of fatty degeneration, focal necrosis and hepatitis have been found at autopsy. The severe toxemia doubtless plays an important part in their production. Indeed, some authors have advanced the theory that death in crisis results in the main from hepatic insufficiency. While no constant cardiac lesions characteristic of crisis have been described, it is abundantly evident to anyone who has witnessed the course of a typical crisis that the heart is struggling against almost insuperable odds under tremendous strain. Indeed, the matter of life or death in thyroid crisis seems to be dependent upon the ability of the heart to hold out against an overwhelming toxemia.

HYPERTHYROIDISM AND PREGNANCY

Since there is a distinct interrelationship between certain of the endocrine glands, it is not surprising that hyperthyroidism and pregnancy should influence each other. Incidentally we know that atrophy and destruction of the pituitary gland results in amenorrhea and sterility. Similarly partial removal of the gland in young animals results in failure of sex development with absence of ovulation and resultant sterility. Since it has long been observed that in pregnancy there is a physiological activation and increased metabolism of the body tissues generally and a heightened activity of the endocrine glands in particular, it follows that patients during pregnancy might present symptoms of increased thyroid activity such as tremor, nervousness, tachycardia and mild emotionalism. The thyroid gland enlarges not only during pregnancy but also during menstrual periods at which time symptoms of irritability, vasomotor instability, tachycardia and tremor are rather common.

In the routine of history taking, we frequently hear that the patient was perfectly well till her first pregnancy during the course of which she developed definite evidences and symptoms of hyperthyroidism that progressed after delivery and led directly into one of the frank types of thyroid pathology such as exophthalmic goiter. Furthermore, the history might indicate that with each succeeding pregnancy, a progressive increase in the hyperthyroidism occurred. Again, in the case of adenoma, a small lump was possibly first noticed during or after the first pregnancy or a pre-existing adenoma may have enlarged or become activated. The activity may have subsided during the puerperium only to be reestablished during subsequent pregnancies. These manifestations explain the "nervous

breakdowns" following repeated pregnancies. The story may repeat itself many times with a gradual increase in size of the original adenoma or the appearance of new nodules. Indeed in certain cases, the dictum "for every child a tooth" might well be paraphrased "for every child an adenoma."

Though pregnancy in the presence of a moderately active exophthalmic goiter is not an unusual occurrence it is rather uncommon in the course of severe exophthalmic goiter. The thyroid toxemia seemingly has a very profound effect upon the pelvic organs. In the early stages, dysmenorrhea and menorrhagia are quite common, possibly as the result of a stimulating effect. In the extremely severe states, a destructive action takes place upon the ovarian and uterine functions resulting in failure of ovulation, long periods of amenorrhea and sterility. These are undoubtedly in the nature of a protective mechanism and indeed in certain instances may actually be life saving.

Because it is clear that pregnancy frequently brings a smoldering hyperthyroidism to light and usually exacerbates an existing one, the question arises: How shall we treat a patient that presents both pregnancy and hyperthyroidism? Previous to 1921, we had no rigid, firm convictions. There is a general notion among physicians that it is dangerous to allow pregnancy to proceed in the presence of hyperthyroidism both because of the possible danger of miscarriage and because of the exacerbating effect of the pregnancy on the hyperthyroidism. Consequently in the past, abortion has frequently been recommended.

In view of our experience since 1921, we are convinced that operation for the relief of hyperthyroidism is entirely feasible and safe particularly in the early months of pregnancy regardless of whether one is dealing with Graves' disease or active adenoma.[8] The patient is promptly relieved of her hyperthyroidism, the worry of both patient and physician regarding the later stages of pregnancy is relieved and normal delivery with a healthy baby is secured at term.

We do not wish to appear dogmatic and convey the impression that we are advising operation upon all patients with goiters complicated by pregnancy. Many of the patients with mild hyperplasias will develop slightly increased nervousness, a mild tremor and tachycardia. These symptoms are amenable to ordinary symptomatic and sedative treatment and hence the patient can be successfully carried through labor and the puerperium particularly if she is

not a primipara. However, when doubt exists especially in the face of increasing activity, the wisest course even in the milder cases would be to submit the patient to thyroidectomy.

When dealing with patients with nodular adenomatous goiters that are often degenerated or cystic and hence show little activity without great increase in the basal metabolic rate, we feel that the pregnancy has little, if any, bearing upon the relatively inactive tumor and operation should obviously be deferred to the puerperium. It is only the active, unmistakable cases of hyperthyroidism that give us concern.

Our early cases, especially those from 1929 to 1939 inclusive, were studied in conjunction with Dr. Alfred C. Beck in the Surgical and Obstetrical Divisions of the Long Island College Hospital. We consider the first four months of pregnancy as the most favorable time for operation since the placental attachment is said to be less firm after the fourth month. Formerly our anesthetic of choice was nitrous oxide-oxygen supplemented by small amounts of ether in order to minimize the danger to the gravid uterus of anoxemia from gas-oxygen alone. The thyroidectomy was carried out expeditiously under customary routine. Morphine was given fairly liberally following operation to prevent uterine contractions. Our results were uniformly satisfactory. The postoperative course was generally free from disturbing nausea and vomiting and devoid of any symptoms referable to the uterus and after twenty-four to forty-eight hours, convalescence was in every respect like that of the ordinary, non-pregnant, hyperthyroid patient.

After the fall of 1935, we replaced nitrous oxide-oxygen-ether by cyclopropane[9] as the anesthetic of choice in the surgery of the thyroid. Formerly there were certain objections to nitrous oxide—possibly theoretical—on the grounds that the unavoidable anoxemia predisposed to fetal asphyxia and abortion. While our results with nitrous oxide failed to bear out this supposed danger, we feel that cyclopropane has all the virtues and none of the disadvantages of nitrous oxide. The high oxygen concentration apparently dispels even the remotest danger of fetal asphyxiation and abortion.

Since 1921, we have taken a firm stand against abortion when pregnancy has complicated hyperthyroidism. We feel that emptying the uterus is generally more of an ordeal and fraught with more trauma and greater danger than an expeditious thyroidectomy. Besides, we are convinced that an induced abortion particularly in

the advanced case is a greater factor in stimulating an accompanying hyperthyroidism than pregnancy itself. After an adequate thyroidectomy, the hyperthyroidism is promptly eliminated and an exacerbation is obviously no longer possible. In an attempt to reduce the problem to rule, we would say that when the question of interruption presents itself, it is the hyperthyroidism and not the pregnancy that should be interrupted.

From 1929 to 1939 inclusive, we performed thyroidectomy for exophthalmic goiter complicated by pregnancy nine times—twice at two and one-half months, once at three months, twice at four months, twice at four and one-half months, once at five months and once at six and one-half months. In one instance in a four month's pregnancy there had been some mild spotting before operation. During the course in the hospital, the patient was observed carefully by the Department of Obstetrics and was delivered at term of a normal baby. The other patients—eight in number—were delivered of normal babies at term. Thyroidectomy was performed on two patients with adenoma complicated by pregnancy—one of two month's and one of three month's duration. The pregnancies eventuated in normal deliveries and normal babies.

We have no exact figures in the cases in which the patients with hyperthyroidism and pregnancy were treated conservatively and in which because of the mild hyperthyroidism, thyroidectomy was postponed to the puerperium. They doubtless were considerably in excess of eleven.

DIABETES AND HYPERTHYROIDISM

Disturbances of carbohydrate metabolism as mentioned are not uncommon in hyperthyroidism. It is, therefore, not surprising that transient glycosuria and hyperglycemia are occasionally noticed particularly in cases of marked severity. They commonly disappear when the hyperthyroidism is controlled. True diabetes on the other hand is relatively infrequent in association with hyperthyroidism. Thus in 4900 cases of hyperthyroidism treated surgically, there were only twenty-five instances of true diabetes—a ratio of 1:196. Of these twenty-five cases ranging in ages from 26 to 67, there were eleven cases of exophthalmic goiter and fourteen of adenoma. These data are at variance with the preponderance of hyperplastic goiter over adenoma reported from other sources. In all of our cases, the diabetes was adequately controlled by diet and insulin and the

prognosis with regard to care of the hyperthyroidism by operation was favorable. Moreover, carbohydrate tolerance was improved following early interruption of the hyperthyroidism. Hyperthyroidism undoubtedly influences diabetes unfavorably and vice versa. Careful blood sugar studies and a careful differential diagnosis between glycosuria and actual diabetes must be made lest one conclude that diabetes has been cured by thyroidectomy.

IODINE AND THE THYROID

Historical. For many centuries it has been known that the ashes of sponges and sea-weed had a beneficial effect upon certain types of goiter. It was, however, not appreciated that the therapeutic effect was due to the iodine content of the ashes. In 1820, Coindet published his successes with iodine in Switzerland. In 1863, Trousseau intending to write a prescription for tincture of digitalis, by mistake prescribed fifteen to twenty minims of tincture of iodine to a patient very ill with Graves' disease. To his very agreeable surprise he noticed after two weeks what we now realize was an iodine remission. He then altered the prescription to one for tincture of digitalis and the patient soon relapsed into her former critical condition. Trousseau, however, was not long deceived and warned against iodine as a therapeutic measure in the treatment of exophthalmic goiter. An enormous and in fact, confusing literature has accumulated since Trousseau's time. In 1896, Baumann demonstrated that the thyroid gland normally contains iodine. Later he and his colleagues showed that iodine given to animals is stored in the thyroid and that when there is a deficiency of iodine in the diet, a compensatory colloid hypertrophy occurs. Kocher, in 1907, while realizing the benefits in endemic colloid goiter treated with iodine, warned against the use of iodine in any and all goiters showing activity for fear of serious consequences. His original warning would seem to be equally apropos today.

Between 1908 and 1911, Marine with Williams and Lenhart demonstrated a definite relationship between the iodine content and the histological structure of the thyroid. They showed that cellular hyperplasia and hypertrophy are due to a deficiency of iodine and that in general the iodine store in the thyroid gland varies directly with the amount of stainable colloid. Kendall, in 1914, isolated the crystalline, active principle of the thyroid and named in thyroxin.

Loevy and Zondek, in 1921, showed that the increased metabolic rate in exophthalmic goiter could be markedly reduced by the administration of potassium iodide. Moreover they observed a striking, though transient, improvement in the clinical symptoms. The thyroid gland has an extraordinary affinity for iodine and the amount that can be stored by any given thyroid varies with the degree of active hyperplasia.

Iodine in Exophthalmic Goiter. In 1924, Plummer published his data on the preoperative administration of Lugol's solution to patients with exophthalmic goiter. The results were startling. He found, and it has since been abundantly verified, that when iodine is administered to a patient previously untreated, in the course of ten days to two weeks a marked clinical improvement occurs. The weakness, nervousness and general status are greatly benefited. Moreover, a significant gain in weight of four to eight pounds or more generally follows. Parallelling the marked clinical improvement, the pulse and the basal metabolic rates fall correspondingly. Indeed in some cases the basal metabolic rates may drop to normal levels, and the patient may well wonder why an operation is necessary. Locally, the thyroid gland commonly swells, becomes firm and even hard, and there is an increase in its density due to a rapid accumulation of colloid in the glandular acini. The vascularity diminishes and the thrills and bruits may be greatly lessened and occasionally actually disappear. It is interesting to speculate as to how the profound changes are brought about. The reduced activity may possibly in some manner fail to demand an increased vascularity. Increased tension on the unyielding thyroid capsule occasioned by the accumulation of colloid within the gland may collapse the vascular spaces and actually force the blood out of the gland. The patient may notice an increase in the size of his neck and complain of a sense of fulness, choking or a mild dysphagia.

Concomitant with the macroscopic changes, there are microscopic changes that are equally striking. The papillomatous infolding of the acini composed of tall, columnar cells with large vesicular nuclei and containing limited amounts of colloid show a complete metamorphosis to the so-called resting stage characterized by large spherical or spheroidal acinar spaces lined by low cuboidal or flattened epithelium with small nuclei. Incidentally the mitochondria which are so indicative of cellular activity are diminished in number and size and have a reduced staining affinity.

It is precisely during the resting stage just described that the thyroid activity is greatly reduced and operation can be undertaken with the least possible risk. Unfortunately this quiescent stage is only transient and in several weeks the secretory activity of the thyroid is gradually resumed and eventually actually accelerated. The gland shrinks, the vascularity increases and in four to six weeks after the iodine is discontinued, there may very likely be an exacerbation of the disease. This condition was known even in the days of Kocher who referred to it as "Jod-Basedow"—an exophthalmic goiter accentuated through the use of iodine rather than one showing progressive activity in the normal course of the disease. Similarly in dealing with patients who, having arrived at the stage of remission just mentioned, were for various reasons allowed to continue under iodine medication, we find that after several weeks the condition will show no further improvement. On the contrary, a chronic iodine phase results during which the striking benefits noticed after two weeks of treatment are gradually lost. The feeling of well-being on the part of the patient slowly disappears. The pulse and metabolic rates gradually rise and the patient may gradually feel herself failing. The true picture becomes masked and the continued use of iodine is without benefit to the patient. If surgical operation is attempted, it is fraught with greater danger than during the quiescent resting period. Indeed if operation is attempted, stage procedures might in the interest of safety become imperative. In either of the circumstances just outlined, the favorable opportunity for operative treatment has been missed and the surgeon will find his judgment severely taxed in both instances.

Iodine and Adenoma. When dealing with adenomas, we should keep foremost in our minds that we are dealing with benign tumors of epithelial origin. There is no evidence to show that the appearance and growth of adenomas is in any way involved in iodine metabolism. Furthermore, there is no evidence to show that an adenoma of the thyroid was ever made to disappear under iodine treatment. Why then should one ever administer iodine for the cure of an adenoma? True, there are some who say they have never seen any harm done to an adenoma by iodine. Parenthetically, we dare say that there is no evidence to show that iodine ever harmed a lipoma, yet few people would prescribe iodine for a lipoma on the grounds that they never saw any harm come from such treatment. Negative statements regarding the treatment of adenoma with iodine are hardly enlighten-

ing and may actually be harmful because of their implications. There is a difference of opinion as to whether or not iodine may convert a non-toxic adenoma into a toxic one. However, we believe that iodine never permanently benefits an adenoma. Furthermore our experience leads up to believe that iodine may sometimes be a factor in causing a non-toxic adenoma to become toxic.

Cutler and Graham in a limited series of untreated cases of clinically active adenomas found that improvement was rather the rule than the exception following the administration of iodine. Moreover, they reported an appreciable recession of the basal metabolism. We are of the opinion that in dealing with adenomas, remissions in the hyperthyroidism following the use of iodine usually indicate the presence in the thyroid of hyperplastic cellular elements comparable to those in Graves' disease. Our first five to six hundred patients with adenomatous goiter were operated upon before the standardized routine preoperative treatment with iodine was generally in vogue. Our results were quite satisfactory. In fact our records since the advent of iodine medication have been no more satisfactory. We are, therefore, indifferent as to whether we do or do not prescribe iodine preoperatively in all cases of non-toxic adenoma. When we give iodine, we do so principally on the grounds that while a brief preoperative iodinization may do some good, it can do no harm unless treatment is prolonged. In the case of undoubted toxic adenoma, we are inclined to agree with Cutler and Graham. Lugol's solution is prescribed for a brief period not to exceed ten days to two weeks following which varying degrees of clinical improvement are occasionally noted.

We believe that the continuous intermittent treatment with iodine of exophthalmic goiter patients over varying periods is fraught with danger[10] and have repeatedly noticed a more satisfactory response of the patient who has never been given iodine as compared to that of the patient who has been treated for some time, possibly intermittently, regardless, too, of the degree or duration of the disease and the dose of iodine. Consequently, we believe that we can predict a striking improvement in the case of the patient who is having iodine for the first time. Conversely, if the patient has had iodine in the past, we can confidently predict a less satisfactory response to further treatment. Some observers believe that failure of improvement under continued treatment with iodine in Graves' disease merely indicates failure to favorably alter the course of the

disease and imply that without iodine, the course would have been more severe. Consequently they continue to prescribe iodine claiming that with time and patience on the part of both physician and patient, the disease will eventually regress. Our experience, and we believe that of most thyroid surgeons, is in disagreement with this view.

When the short intensive preoperative iodinization of the patient has not been carried out and she comes to us partially, incompletely or intermittently iodinized, we are confronted with a situation in which the basic expression of the disease is masked. Such a case must be given individual attention. No definite routine seems applicable and satisfactory. Sometimes we interrupt the iodine treatment and sustain the patient with sedative and symptomatic treatment for six to ten weeks to allow the thyroid to eliminate the excess of iodine and to become sensitive again to iodine. Lugol's solution is then given for ten days to two weeks. Operation can then be undertaken under more favorable conditions. In other instances, to conserve what gains might have been made, we continue the use of iodine in increased dosage augmented possibly by several daily intravenous injections of sodium iodide for a period of a week or ten days. If the condition improves appreciably, operation is performed. If no improvement or on the contrary intensification of the clinical symptoms occurs, the iodine is discontinued and operation postponed for six to ten weeks. While the second iodinization usually improves the patient, the amelioration of symptoms is usually less striking than following the standardized first period of treatment. A certain number of cases may become refractive to iodine.

Thus far we have been discussing the role of iodine in hyperthyroid states. While our subject matter is that of hyperthyroidism, it might not be unwarranted to mention briefly the only type of goiter in which in our opinion iodine is indicated for therapeutic cure—simple colloid endemic goiter, the inactive type of parenchymatous goiter commonly developing when there is a deficient intake or a faulty metabolism of iodine. It occurs endemically in so-called goiter belts throughout the world. It may be prevented by the prophylactic administration of minute amounts of iodine. Colloid goiter does not produce hyperthyroidism but frequently shows varying degrees of hypothyroidism. Small doses of thyroid extract as well as iodine are then indicated. A caution should be sounded lest overtreatment with iodine stimulate the gland and superimpose a state of hyperthyroidism.

GENERAL TREATMENT

Puberty Hyperplasia. In our discussion of the mild hyperplasias of the puberty type we indicated that they may undergo spontaneous cure. Medical treatment consists principally of mental and physical rest and the administration of tonics and sedatives. A careful routine of eating, sleeping and physical activity should be instituted. A young girl may have to forego her schooling or employment on account of nervousness and emotional instability. Hence the economic loss may be considerable. Since *x*-rays inhibit hyperplastic tissue, a series of mild *x*-ray treatments may occasionally be a helpful adjunct to medical treatment. Inordinate doses, however, should be mentioned only to be condemned. Relapses after apparent cures are common and it is not surprising that a goodly number of the milder hyperplasias progress to the more moderate types and that a certain number may eventuate in exophthalmic goiter. Detailed histories indicate that the progression is not too uncommon and may be facilitated by the increasing strain of life as the young girl becomes older and particularly when the responsibilities of marriage and childbearing enter her life. Iodine should not be included in the medical treatment lest it produce an exacerbation or conversion into a more active type of hyperplasia. If medical treatment fails, recourse to operation may become necessary.

Exophthalmic Goiter. In dealing with patients presenting exophthalmic goiter, one must always bear in mind their extremely sensitive, high-strung and complex natures. All sources of anxiety, fear and worry should be eliminated. A sympathetic approach on the part of nurses and doctor is necessary to gain the patient's confidence. The routine of life should be carefully controlled—admittedly a difficult task in the case of those who by dint of economic resources are forced to earn their living by jobs that may strain their already overtaxed nervous systems. If the economic status permits, a vacation under a complete change of environment and freedom from responsibility may be of untold benefit. General measures including sedative treatment, an adequate balanced diet stressing carbohydrates and a maintainance of the water balance should be instituted. In brief, the medical management consists of rest—both mental and physical—and general supportive measures. Exophthalmic goiter is a progressive disease and should be treated surgically when frankly expressed. In the early cases, operation is indicated if a period of three to four

months of rest and medical treatment fail to halt the condition. Because they tolerate suspense poorly, most patients once convinced that medical treatment is no longer helpful accept operation graciously. In the highly emotional and fearful, we have found that a brief period of hospitalization in or in close proximity to a ward containing convalescent postoperative cases of hyperthyroidism will work wonders in preparing the prospective patient psychologically and emotionally for the ordeal of operation. When operation is finally agreed upon, Lugol's solution, which during the preliminary symptomatic and medical treatment had been purposely withheld, is given in doses of M X—XV t.i.d. and after ten to fourteen days a striking remission as described under "Iodine and the Thyroid" is customarily obtained. Operation should be undertaken during the period of remission.

Adenoma. In dealing with toxic adenomas of the thyroid the problem is comparatively simple. We are dealing with tumors that per se are causing the hyperthyroidism. Surgical removal of all the adenomatous tissue is, therefore, the rational treatment. Prophylactic removal of non-toxic adenomas is advisable to prevent them from becoming toxic, to avoid future pressure effects and to forestall malignant degeneration which occurs in 2 to 4 per cent of cases.

Hyperthyroidism with Degenerative Effects. The Thyrocardiac. We have already detailed the severe consequences to the myocardium resulting from continued hyperthyroidism. These may express themselves in a variety of ways and add greatly to the difficulties of the preoperative preparation. The cardiac manifestations may overshadow and mask the hyperthyroidism. The incidence of heart failure is generally higher with toxic adenoma than with Graves' disease. This may be due to the greater chronicity of the toxemia resulting from adenoma as well as preexisting cardiac disease—arteriosclerosis, hypertension or depleted circulatory reserve—incidental to the average greater age of the patients afflicted with adenoma.

The preoperative preparation of the thyrocardiac is extremely important and demands patient and thoughtful consideration. Rest and sedatives are necessary in every case. Rest in bed may be imperative especially when there are evidences of decompensation. Digitalis in adequate dosage is indicated. If edema supervenes, fluids should be restricted to aid in the restoration of competency. In selected cases presenting severe peripheral edema and abdominal ascites, we have

occasionally resorted to intravenous diuretics such as mercupurin or salyrgan in conservative doses. They should, if used at all, be given with extreme caution and are contraindicated when renal function is imparied. Every effort should be made to obtain cardiac compensation since it is ordinarily not wise to undertake thyroidectomy before all evidences of decompensation are overcome. When competency of the circulation has been established, we are prone to wait a few days or longer to see how well competency is maintained or if the heart again becomes incompetent after the fluid intake is increased or the diet is liberalized. When maximum improvement has been obtained and maintained, the intensive preoperative treatment with Lugol's solution is commenced and further striking improvement is usually obtained. Thus if fibrillation has been present, the rate tends to decrease and become regular, the cardiac reserve is strengthened and the general physical condition of the patient is enhanced. It is of course realized that it is not always possible to completely restore the normal rhythm. The period of treatment varies in the individual case from four to seven weeks instead of the ten days to two weeks in uncomplicated case. Obviously two weeks is hardly sufficient time to overcome fibrillation, eliminate edema and build up cardiac reserve. We feel that an occasional fatality may follow a too hasty preoperative preparation. Slow preparation allows the patient to consolidate her gains and renders her a much better surgical risk. It provides the surgeon with a better perspective and a finer prognostic view. It should be realized that in the occasional "desperate risk" patient, who perhaps is suffering from general anasarca, extreme dyspnea and even cyanosis, prolonged preparation may be out of the question. Iodine may have to be given promptly and intensively. It may indeed be the patient's only chance of survival.

OPERATIVE TREATMENT

When the maximum clinical improvement has been obtained by preoperative preparation, operation should be undertaken.

Preoperative Medication. Amytal grs. 1½ is given the night before operation. Morphine gr. ⅛ is given one hour before the scheduled time of operation and repeated immediately before the patient is sent to the operating room. When using cyclopropane, too much preliminary medication tending to depress respiration is not advisable since cyclopropane is not a respiratory stimulant.

Anesthesia. Cyclopropane. From 1919 to October, 1935, nitrous oxide and oxygen with or without ether had become the anesthetic of choice in the Goiter Clinic of the Long Island College Hospital. Since then, cyclopropane[9] has practically supplanted all other forms of inhalation anesthesia in our clinic. It is said that cyclopropane has the potency of chloroform and ether without their irritant qualities, and, excepting in high concentrations, is not measurably harmful to liver, kidneys or heart. A high potency may produce spasm of the larynx. The usual laryngeal reflexes are, therefore, not present to warn the anesthetist. Consequently, hasty induction must be avoided. Unlike nitrous oxide and ethylene which increase the rate and volume of respiration and even produce depression from oxygen deficiency, cyclopropane causes comparatively little if any change in the rate and volume of breathing unless excessive concentrations are used. The large amounts of oxygen given with the gas prevent cyanosis and the blood, consequently, should always maintain a bright red color.

Only a few minutes are required to produce surgical anesthesia in the average patient with hyperthyroidism. Unusually nervous patients, especially those with high metabolic rates require a longer induction and greater concentrations of the gas. An initial potency of 5 to 10 per cent of cyclopropane with 90 to 95 per cent of oxygen is generally satisfactory and the operation can usually be continued with the lower limits of concentration. There is no excitement period and the various stages from induction to deep narcosis are merged rapidly. However, the patient is at all times protected by the large concentrations of oxygen. Unlike nitrous oxide and ether, cyclopropane does not produce choking, burning or strangling. It is not unpleasant. Recovery is quick and the patient can usually coöperate promptly. Postoperative nausea and vomiting are less prolonged than with other inhalation anesthetics, notably ether.

In exophthalmic goiter with greatly elevated metabolism, the patient's demand for oxygen is frequently so great that we have in the past had difficulty when employing nitrous oxide in preventing anoxemia. Ether frequently had to be supplemented to obtain relaxation. Venous congestion during the operation was at times disturbing, postoperative nausea and vomiting were increased, the postoperative conscious responses were delayed and acidosis and acetonuria were more frequently encountered.

Inasmuch as a large number of goitrous patients have varying degrees of cardiac damage, a powerful anesthetic like cyclopropane which can be administered without producing anoxemia has distinct advantages. Most thyrocardiac patients require more than ordinary amounts of oxygen. When cyclopropane is used, the high oxygen ratios tend to protect the overburdened myocardium against the strain of operation.

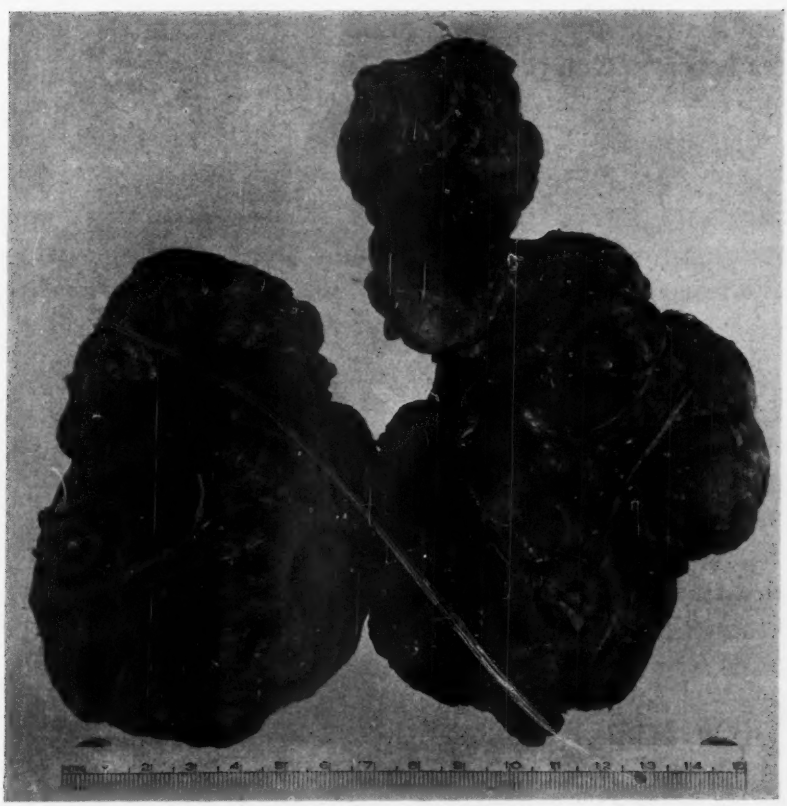
In removing large adenomatous goiters, particularly those that have caused tracheal narrowing or compression, cyclopropane is especially helpful. The tracheal reflexes are quickly abolished and sufficient oxygen can usually be supplied to prevent respiratory distress, venous congestion and asphyxia, even with passive breathing. The quick abolition of the throat reflexes by cyclopropane is of great value when recourse to airways becomes necessary. Helium is a valuable adjunct in many such cases. It has been demonstrated experimentally that cyclopropane in concentrations sufficient to produce surgical anesthesia does not seriously depress liver and renal function. It is, therefore, well suited for cases of prolonged hyperthyroidism with hepatic and renal damage.

Avertin. In cases of extreme toxicity in which emotionalism, great anxiety, fears and phobias are the outstanding symptoms, we have given basal avertin anesthesia to great advantage as a preliminary to cyclopropane. Following doses of from 50 to 80 mg. per kilo of body weight, the patient comes to the operating room greatly relaxed both mentally and physically—in a kind of twilight sleep. The induction and the operative course are consequently greatly facilitated.

Local Anesthesia—Novocaine. Occasionally when dealing with large adenomatous goiters we have used local infiltration with ½ to 1 per cent novocaine. If compression of the trachea threatens, the operator with the patient's coöperation can usually manipulate the tumor mass so as to avoid strangulation. The laryngeal reflexes are maintained. The fear and trepidation of the patient is counteracted by indulging him in light and carefree conversation. Local anesthesia may be enhanced by repeated small hypodermic doses of morphine during the course of operation or supplemented by cyclopropane, or other general anesthetic.

Intratracheal Anesthesia. In certain cases of large adenomatous goiters with tracheal compression, especially if they are largely intrathoracic, it is difficult even with cyclopropane to supply enough

A B



C

FIG. 7. R. G., age 57. A, huge bilateral adenomatous goiter which required intratracheal anesthesia. B, postoperative photo taken three weeks following operation. C, photo of gross specimen.

oxygen. The delivery of the mass from the thorax may further constrict the already compromised trachea. In the exceptional case, the introduction of an intratracheal catheter as a preliminary to operation will solve the difficulty. (Figs. 7A, B and C.)

TECHNIQUE OF OPERATION

Exophthalmic Goiter. The operation of thyroidectomy has in a large measure become well standardized. Obviously, however, in view of the marked variation in the size, position and clinical activity of a goiter one cannot follow a rigid procedure. It is our purpose, therefore, to outline the principal steps in our routine and to refer the reader to standard textbooks and monographs for anatomical and other details.

Because the vast majority of patients are women, the postoperative scar becomes a matter of compelling interest. The collar incision is universally employed. It should follow and parallel the folds of the skin at a sufficiently high level to place the ultimate scar at the best cosmetic level. The upper and lower flaps are thoroughly mobilized. Bleeding is minimal if the exact line of cleavage is maintained. A self-retaining retractor is used to spread the upper and lower flaps. The ribbon muscles are freed from the thyroid by sharp dissection and separated from the gland by a retractor held by an assistant. When the gland is inordinately large or has an unusually high superior pole, the prethyroid muscles may be divided to facilitate exposure. However, we find no need for routine division and when resorted to, make the division at a high level to conserve the innervation and retain the normal muscle tone.

The right lobe and isthmus are separated from the left lobe by careful dissection over the midline. When a prominent isthmus is present or the trachea is deeply situated, separation is greatly facilitated by a puncture wound of the knife in the suspensory ligament through which a curved Kocher clamp is carefully guided along the tracheal fascia to the lower limits of the juncture of the right and left lobe. By a careful, light spreading of the clamp, a practically bloodless groove is obtained and separation of the overlying isthmus from the left lobe is easily accomplished. The lobe is further freed by careful dissection medially along the trachea; the handle of the scalpel is used to exert firm but gently pressure to gain further mobilization. Next, attention is directed to the upper pole which on account of its fixation, may be difficult to free. However, if the sus-

pensory ligament is severed, a relatively free space may be developed between the medial aspects of the upper pole and the cricoid and laryngeal cartilages. By carefully holding a Kelly clamp in close proximity to the thyroid capsule and gently spreading it in the axis of the tracheolaryngeal groove, some fibrofatty supporting tissue is easily severed and a bloodless separation is obtained. The superior pole and superior thyroid artery are lifted to the side and well away from their proximity to the superior laryngeal nerve which among other things supplies sensory filaments to the epiglottis, base of tongue and the mucous membrane of the larynx as well as motor twigs to the cricothyroid and, according to some authors, to the interarytenoids which are concerned in the approximation of the posterior portion of the vocal cords. The upper pole is mobilized posterolaterally by gently freeing any muscle or fascial attachments with the handle of the knife or a dissector of the type of the Küttner clamp. The superior thyroid artery is clamped, divided and separated from the gland. If the preceding steps have been adequately performed, the pole can be easily lifted and rolled upward and inward. Forceful traction against the trachea and larynx should be avoided. The artery can now be ligated without fear of injury to the superior laryngeal nerve. Voice changes and irritative postoperative coughing formerly attributed solely to recurrent nerve injury will be further reduced by protecting the superior laryngeal nerve.

The lower pole is gently lifted and the lobe is rotated and gently freed from any posterolateral attachments. Dividing and transfixing of the inferior thyroid and capsular veins further facilitate mobilization. As the gland is elevated and separated from any supporting fibrofat posteriorly, great care must be exercised to avoid the parathyroids. Unless adequate mobilization is obtained, the operator has no real way of estimating the amount of tissue to remove. Therefore, before commencing the resection he should make certain that both poles are elevated and that he has discovered any posterior projections or extensions of tissue. Having decided upon the amount of tissue to remove, the operator may commence either laterally or medially, placing a row of guide and hemostatic clamps along the capsules at a level above which he decides to commence the resection. The medial row should be well above the tracheo-esophageal groove particularly in the vicinity of the tracheolaryngeal angle where the superior parathyroid body is especially prone to be injured. The outer row must be placed lateral to the entrance of the inferior

thyroid artery and run in a slightly upward curve toward the beginning of the anterolateral branch of the superior thyroid artery. The exact level of the row of clamps in the individual case is determined by the operator's estimate of the amount of tissue he wishes to remove. When less radical resections are desired, the clamps are placed correspondingly higher. By properly placing the guide clamps the posterior "danger zone" containing the parathyroids and the recurrent nerve is avoided. Therefore, tetany and recurrent nerve injuries should virtually never occur. Resection is carried out by wedge removal, the apex of the wedge being directed toward the posterior capsule. Bleeding points are clamped as they are encountered. We have not found it necessary to ligate the inferior artery as a preliminary to resection and are inclined to believe that routine ligation of this artery might increase the tendency to parathyroid tetany since the sole blood supply of the parathyroids comes directly from the inferior thyroid arteries.

The lateral and medial capsules are approximated by a running fine catgut suture and a series of interrupted fine silk sutures. Unless a stage procedure has been decided upon, the left lobe is exposed and resected. The amount of tissue to be removed is estimated to some extent on the size of the right remnant. The operative field is closely examined for any minor bleeding and when a dry field has been obtained, closure is undertaken. The ribbon muscles are united over the midline by small interrupted silk or fine catgut sutures. Their lower limits are not approximated so as to allow any subsequent collection of blood and serum in the depths to come to the surface. A row of fine interrupted silk sutures about 1 cm. apart are used to unite the subcutaneous flaps. Finally, a series of fine silk sutures are employed to coapt the skin.

Should the wound be drained after an operation for goiter? When dealing with small firm glands in which a dry field is obtained with relative ease, drainage is not necessary. However, when dealing with large, soft and vascular glands requiring much suturing to control hemostasis, a small tissue or cigaret drain is inserted under the upper flap between two skin sutures and removed in the evening of the day of the operation.

Adenomas of the Thyroid. In dealing with nodular or adenomatous goiter, the problem resolves itself into removing all adenomatous nodules and leaving all the healthy tissue in order to maintain normal thyroid function. Most of the principles and procedures already de-

scribed are applicable. However, on account of the frequent distortion of the surgical anatomy as regards the thyroid arteries, parathyroids, laryngeal nerves and the trachea, the customary land-

A

FIG. 8. M. O., age 50. A, *x*-ray film showing large intrathoracic adenoma with pressure effects. B, photo taken three weeks following sternotomy for removal of intrathoracic goiter.

marks that guide the operator may be greatly obscured and unusual precautions may be necessary to avoid operative and postoperative complications. Moreover, transverse division of the prethyroid muscles will have to be practiced with greater frequency than in dealing with hyperplastic goiters. Ample mobility must be obtained lest one or more nodules escape detection and in later years cause recurrent hyperthyroidism. A row of clamps is placed along the medial and lateral capsules at a level well above the posterior "danger zone." Since adenomas have discrete capsules, a plane of cleavage

can usually be developed by making an incision to the depth of the nodule and shelling it out from the surrounding thyroid tissue. Where a number of nodules are present, they should be enucleated seriatim.

B

FIG. 8. For descriptive legend see opposite page.

In dealing with multiple small nodules particularly when the intervening thyroid tissue is hyperplastic, resection-enucleation is practiced. To facilitate resection-enucleation, the anterior surface of the lobe which is usually thinned out and frequently fibrous or degenerated, may be excised. By this unroofing process, access to the underlying pathology is obtained and an intracapsular wedging procedure is carried out as radically as necessary to remove all adenomatous nodules and the bulk of the hyperplastic tissue. The most healthy looking tissue will generally be found along the posterior capsule about the upper pole and sufficient tissue can nearly always be salvaged to prevent postoperative hypofunction.

In dealing with substernal goiter, complete mobility and luxation are of paramount importance. To gain ample mobility, the prethyroid muscles will almost certainly need transverse division. It is important to free the mass along the trachea. All division of tissue

should be made after careful double clamping. The upper pole must be thoroughly mobilized. The suspensory ligament and any fascial attachments are carefully freed and the superior thyroid artery is ligated at a high level. It should be remembered that in the process of descent, an intrathoracic goiter carries downward with it the thyroid arteries and superior, middle and inferior veins. Thus by double clamping and dividing the main blood vessel trunks along the capsule, the circulation can be completely controlled from above. The tumor mass is finally freed and delivered by gentle manipulation and extracapsular dissection along a well-defined plane of cleavage. If the preceding maneuvers have been carefully carried out, it is surprising how readily even large masses can be guided upward into the operative field. When central softening or cystic degeneration has occurred, puncture of the capsule may facilitate delivery. We have had recourse to sternotomy only once and have had to use intratracheal anesthesia in rare instances. (Fig. 8A and B.)

Stage Operations. One of the most important principles guiding the thyroid surgeon is that the operative procedure should not go beyond the limits of the patient's tolerance. Fortunately a single complete operation that might prove fatal can generally be carried out successfully in successive stages; the extent of the surgical procedure and the number of stages in each instance depend upon the individual case. Whether one or two preliminary ligations should precede a single lobectomy followed later by a second lobectomy will depend upon the condition of the patient when first seen before the real status of the hyperthyroidism is masked by suppurative treatment—rest, fluids and cardiac support. The surgeon's judgment will frequently be severely taxed. There is unfortunately no substitute for experience, nor can any rigid rules be given. However, when in doubt, one should not hesitate to do stage operations for it is obviously wiser to operate two or even three times safely than to have a complete operation and a dead patient.

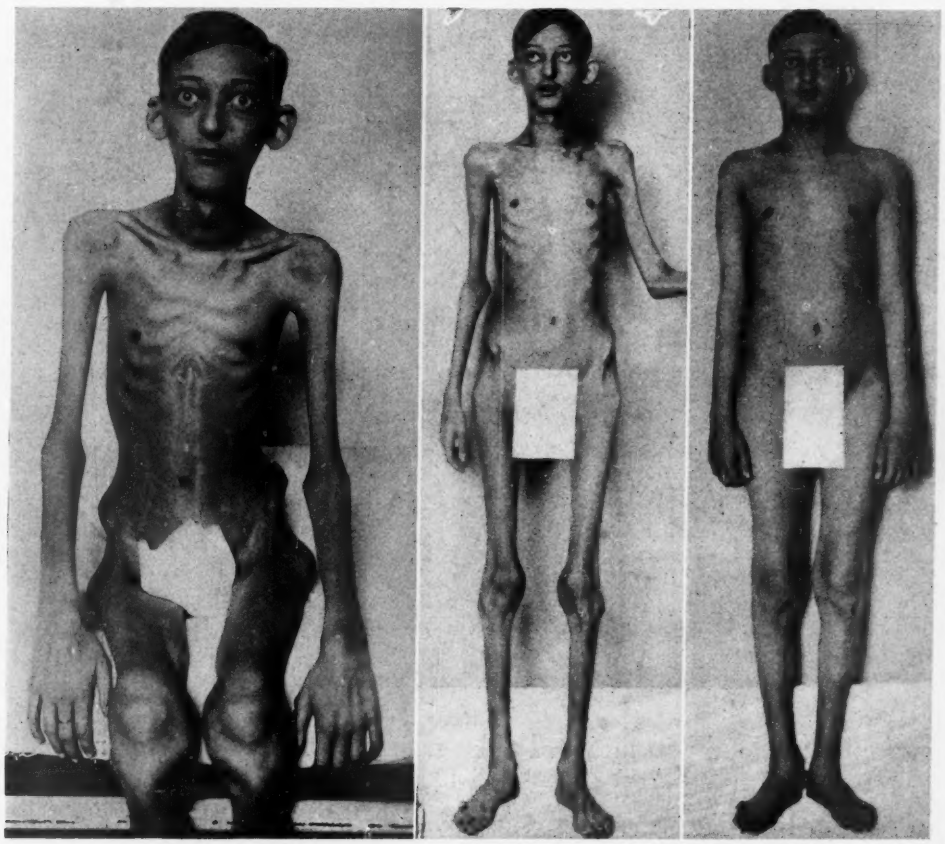
Preoperative Criteria of Operability. In attacking the problem of operability, experience has shown that certain preoperative factors indicate the wisdom of stage procedures in safeguarding mortality. Among these are inordinate weight loss, severe asthenia, prolonged severe hyperthyroidism especially in the presence of marked cardiac damage, the presence of chronic valvular disease, inordinately high metabolic rates especially when maintained in spite of adequate preoperative iodinization, vomiting and diarrhea and unusual mental

states bordering on psychosis. The indiscriminate and improper use of iodine may mask the true state of the hyperthyroidism and lead to the necessity for stage procedures. While the correct preoperative use of iodine has, excepting in very unusual instances, obviated preliminary ligations, it has by no means excluded the necessity for stage operations. In dealing with extreme degrees of hyperthyroidism, a single ligation may be done to test the operability. If the patient's reaction is favorable, the operation may be undertaken following the average routine. If the reaction is severe, caution and stage procedures would seem to be in order. (Fig. 9A, B, C and D.)

Operative Criteria. In our own experience it has become abundantly evident that postoperative crisis with which it is difficult to cope remains the principal cause of death. Even though the hyperthyroidism is partially controlled by careful preparation, the basic hypersensitivity of the patient to operative assault may still be present to a marked degree. It is revealed in the emotional reactions of the patient during the immediate preoperative period and just before anesthesia, as well as during the reactions produced by unavoidable trauma associated with the operation. The preoperative manifestations of tachycardia, nervousness, flushing, tremor and throbbing are secondary to the emotional factors of fear and anxiety. Likewise, the operative reactions of increased blood pressure and pulse rate, leukocytosis, increased blood sugar and a rise in temperature are secondary to trauma and other deleterious factors of the operation.

In an attempt to evaluate the operative reactions, it became apparent to us (Goetsch and Ritzmann),[7] that they showed striking resemblances to the reactions produced by the injection of small amounts of adrenalin in patients suffering from hyperthyroidism. Furthermore, the patients that were particularly sensitive to adreanlin as determined by the adrenalin test (Goetsch)[11] invariably showed proportionately sharp postoperative reactions. Even crisis seemed to be an exaggeration of the responses which can be produced by adrenalin in the hyperthyroid patient. The differences were solely of degree rather than type and character. The striking parallelism between the adrenalin and operative reactions suggested strongly that the latter are due to a hypersecretion of adrenalin produced by the factors inherent in the operation—emotion, fear, operative trauma and anesthesia—in stimulating the suprarenal glands. The sympathetic nervous system already rendered hypersensitive by the

hyperthyroidism would appear to be further stimulated by the additional adrenalin. It was moreover found that the postoperative hyperthyroidism is proportional to the degree of the operative reac-

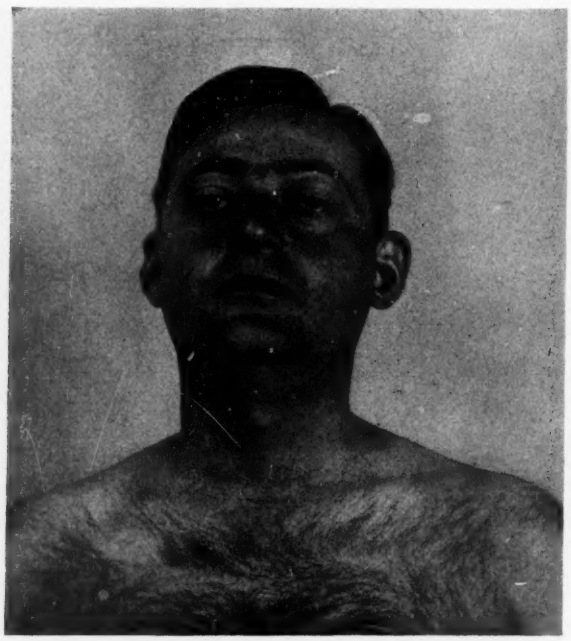


A B C

FIG. 9. H. K. A, age 15. Photo taken 4/9/27. Severe exophthalmic goiter. Patient in extremis from marked emaciation and debilitation. B, age 16. Photo taken 4/19/27 following two previous superior ligations in stages. C, age 16. Photo taken 6/6/27. Note further marked physical improvement following previous superior ligations. D, age 26. Photo taken in October, 1938, ten years following two lobectomies in stages after two individual ligations had been done previously. It required four stage operations to obtain this result.

tions. Thus an early sharp rise in the pulse rate, blood pressure, pulse pressure and respirations should warn the surgeon of an unusual sensitivity and a threatened severe postoperative reaction especially if these factors rise progressively and are sustained. The desirability of restricting the operation and adopting a stage procedure become apparent. Reduced to clinical practicality we would say that given a case of hyperthyroidism that on the basis of preoperative criteria

presents a problem in operability, we have a rather recent and helpful criterion of the patient's tolerance for operation in an evaluation of the operative reactions. If by the time the first lobe has been re-



D

FIG. 9. For descriptive legend see opposite page.

moved, the blood pressure and pulse rate have tended to rise progressively, a first stage single lobectomy had best be done in the interest of safety. In this connection, the behavior of the blood pressure has come to mean more to us than that of the pulse. We believe that careful study and interpretation of the operative reactions may aid in reducing the incidence of severe postoperative hyperthyroid reactions including crisis.[12] (Figs. 10 and 11.)

Amount of Tissue to Be Removed. One of the most important problems in the surgery of goiter with hyperthyroidism is the estimation of the amount of tissue to be removed. Once again no infallible rules can be laid down. In general the younger the patient and the more recent the hyperthyroidism the more radical the resection must be to prevent recurrent hyperplasia. In older patients, the glands tend toward diminished functional activity and less radical resections are indicated to preserve normal physiological thyroid activity. Obviously, when too much tissue is removed an undesirable state of hypothyroidism and even myxedema may result and, conversely, if

too little tissue is removed a persistent postoperative hyperthyroidism may follow. In the individual case an estimate is not based on any one criterion such as the basal metabolic rate. Rather one must

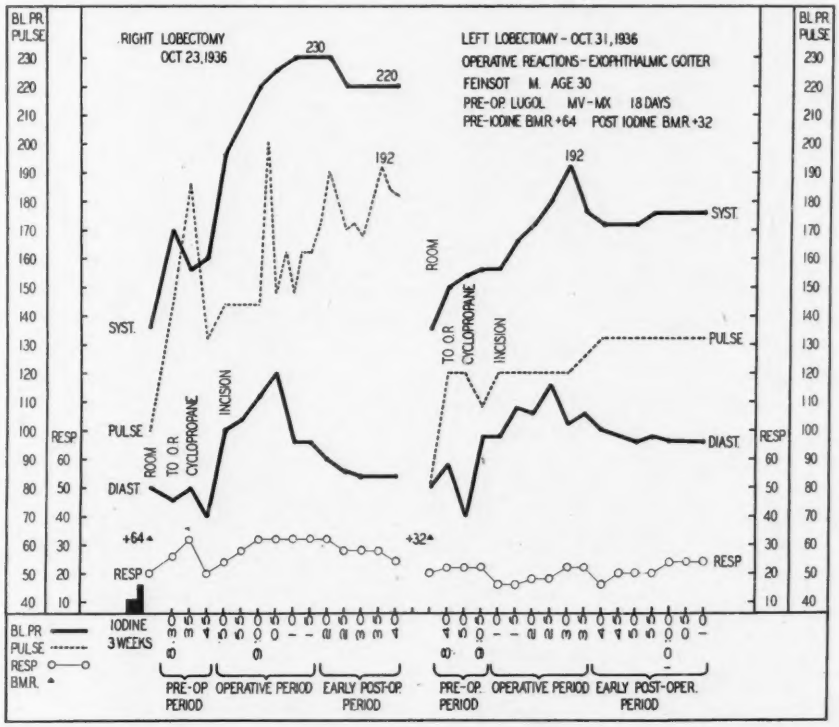
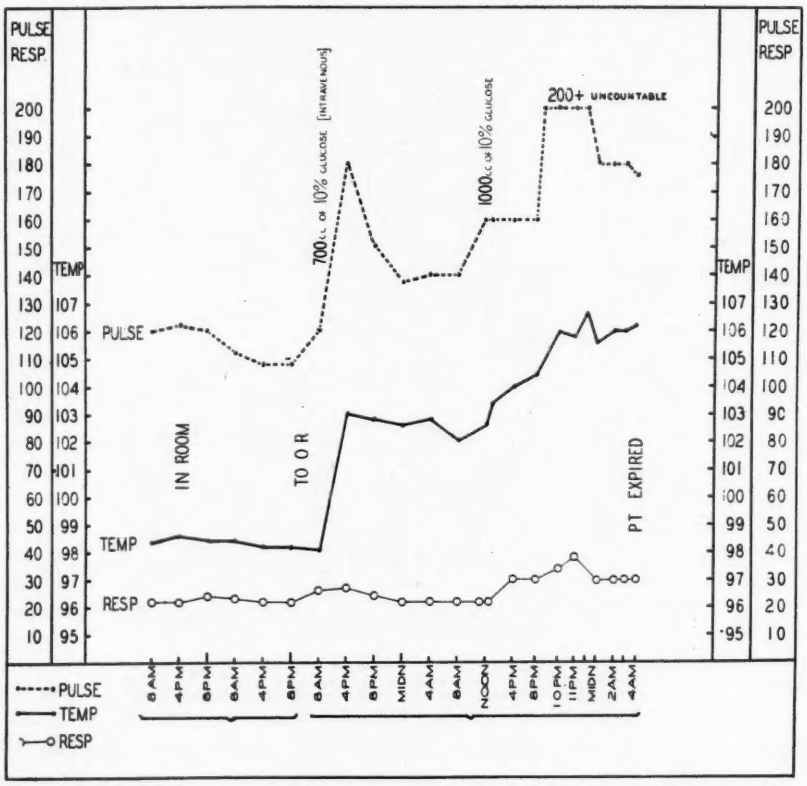


FIG. 10. Severe operative reaction during first stage right lobectomy for marked hyperthyroidism due to Graves' disease, indicating need of restricting the operation. Note continued climb in pulse and blood pressure. Compare with less severe reaction during second stage left lobectomy.

first of all view and interpret the composite picture—the clinical activity as expressed by tremor, tachycardia, loss of weight, the presence or absence of mental deterioration and the basal metabolic rate. Secondly, the individual's reaction to preoperative iodine medication is important. In those patients that react favorably to preoperative treatemnt with iodine, there will be a marked fall in the basal metabolic rate, an appreciable gain in weight, a diminition in the pulse rate and a general improvement in the nervous symptoms. At operation the glands of such patients will invariably show an abundant content of colloid. They will be firm, pale and relatively avascular. Sections are characteristically translucent. During the stage of iodine involution, the gland is relatively inactive and its bulk is mostly inert colloid. Obviously in dealing with such glands the

reduction should not be too radical. However, when the clinical response to iodine is limited and the gland at operation is still very vascular and fleshy in appearance indicating lack of complete involu-


PULSE
RESP
TEMP
700 c.c. of 10% GLUCOSE [INTRAVENOUS]
1000 c.c. of 10% GLUCOSE
200+ UNCOUNTABLE
IN ROOM
TO O R
PT EXPIRED
PULSE
TEMP
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6 AM 4 PM 8 PM 8 AM 4 PM 8 PM 8 AM 4 PM 8 PM MIDN 4 AM 8 AM NOON 4 PM 8 PM 10 PM 11 PM MIDN 2 AM 4 AM

FIG. 11. Severe postoperative reaction ending in death from crises less than forty-eight hours following second stage left lobectomy for severe exophthalmic goiter in a young girl, aged 19. Note the abrupt climb in both temperature and pulse rates. Just before exodus, the pulse was uncountable and the temperature 106°F.

tion, a relatively more radical reduction must be made. In cases presenting marked cardiac damage, radical reductions are in order even at the expense of producing underfunction so as to relieve the overloaded heart.

POSTOPERATIVE COURSE AND TREATMENT

In the average case in which the hyperthyroidism has not been too prolonged or too damaging to the cardiac and physical status of the patient and in which no special operative difficulties have occurred, the patient may confidently be returned to his room and reuuire only the customary, routine symptomatic treatment. If

cyclopropane has been used consciousness is quickly established. There is little danger of inspiring vomitus and the patient may soon be placed in a low Fowler position and later shifted to such positions

FIG. 12. A. C., age 32. Recurrent hyperplasia in pyramidal lobe one year following subtotal resection for exophthalmic goiter.

as are most comfortable. Small repeated doses of morphine are used during the first forty-eight hours to control discomfort and pain. Later bromides and phenobarbital derivatives are usually adequate. Persistent vomiting and dehydration are best controlled by intravenous and rectal administration of glucose solutions according to the rationale elaborated above under the heading of "Acidosis following Operation." Fluids are given by mouth as soon as tolerated and a small bland breakfast is frequently possible and often acceptable on the morning after operation. High caloric foods especially carbohydrates should be encouraged and are usually well tolerated. The peak of the postoperative febrile and pulse reactions occurs in about thirty-six hours and should be controlled with ice caps over the precordium and the head and cold sponging. Heart stimulants are rarely needed. However, during recovery an occasional case may

show a fall in blood pressure and cyanosis requiring caffeine, strychnine or other stimulants. Arhythmias, fibrillation and broken compensation require digitalis and similar medication. The rapid pulse rate is unaffected by digitalis and usually drops spontaneously after the postoperative febrile reaction has spent itself. The skin sutures are removed in forty-eight hours so as to prevent unsightly punctate scarring of the suture holes. During the first twenty-four to forty-eight hours, the house surgeon and nurses should be on guard for complications such as crisis, hemorrhage, respiratory distress, pulmonary edema and tetany.

Crisis. The characteristics of crisis have already been detailed. Early recognition and treatment of this grave complication are imperative. Since crisis may occur with startling suddenness or appear gradually with definite premonitory symptoms such as increased excitability, loss of emotional control bordering on delirium, increasing hyperpyrexia and an alarming rise in the pulse rate, the house surgeon and the nursing staff should be trained to suspect crisis. The extreme restlessness may require unusual measures. Heavy sedation—morphine and other hypnotics and sedatives—may have to be administered liberally. The hyperpyrexia should be combatted with ice caps over the precordium, head and body and by repeated sponging with ice water. Because of the rapid consumption of glycogen in crisis, the glycogen reserve should be fortified by frequent intravenous injections of glucose solutions. The treatment should be aimed at sustaining the heart until the critical reaction has spent itself since death in crisis is invariably due to secondary cardiac failure.

Hemorrhage. Hemorrhage may be severe if each successive step in the operation has not been controlled before proceeding with the next. Factors that are important are (1) adequate exposure, (2) developing and adhering to the normal planes of cleavage, (3) early clamping, division and ligation of the lateral and inferior veins to prevent tearing and retraction, (4) individual ligation of all focal bleeding points, (5) the avoidance of mass ligations and (6) the insistence on a dry field before closing. With the preceding precautions, serious postoperative hemorrhage should be rare. When bleeding does occur, it happens usually during the first twelve to twenty-four postoperative hours. It makes its presence known by a gradual, progressive tenseness of the skin flaps and a gradual increasing respiratory distress. Early reflex coughing increasing to dyspnea and

eventual stridor may follow in rapid succession. No time should be lost in prompt exploration of the operative field and control by suture ligatures of the bleeding points. Incidentally, early interference may prevent the necessity for emergency tracheotomy. In the event that tracheotomy becomes necessary, it should be done as deliberately as circumstances permit and at a low level. If done at a high level in proximity to the cricoid, tracheal stenosis may result since the trachea is narrowest at this point.

Injury to the Recurrent Nerves. Formerly recurrent nerve paralysis was cited as among the most frequent and serious complications in goiter surgery. However, the present day improved technique has reduced serious injury to almost nil and relegated slight injury causing transient paresis and mild horseness to relative infrequency. Adequate mobilization, light manipulation, expeditious delivery of the gland, a wholesome regard for anatomical landmarks, the avoidance of roughness and bleeding along the posterior "danger zone" will do much to prevent nerve injury. Scrupulous care should be excised especially posterolaterally where the nerve is in close proximity to the inferior thyroid artery. A common notion is that the nerve is in greatest danger in the vicinity of the lower pole. However, the most vulnerable point is undoubtedly along the lateral deeper aspects of the cricoid cartilage in relationship to the cricopharyngeal muscle. Care should be exercised in this area lest the guide clamps be placed too low. Moreover, deep, massive suturing to control bleeding which at the cricolaryngeal angle may at times add to the operative difficulties, should be avoided.

Pulmonary Complications. Refinements in the administration of anesthetics, improvements in surgical technique and postoperative management have greatly reduced the incidence of pulmonary complications. Notably the avoidance of recurrent nerve injury and the rapid recovery from cyclopropane have greatly minimized the dangers of bronchitis and aspiration pneumonia. Further factors in prophylaxis have been the general recognition for postponing operation after recent respiratory infections and for thoroughness in the preparation of the thyrocardiac for operation.

Tetany. Tetany is a condition of increased irritability of neuromuscular control due to depletion of the calcium level in the blood. Postoperative tetany usually develops within eighteen to forty-eight hours. It is seen less frequently today than in the early era of thyroid surgery. Tetany may occur after removal or bruising of the para-

thyroid glands or following injury to their blood supply. In mild cases, certain prodromal symptoms may indicate an approaching tetany. Headache, a feeling of weakness, pain and stiffness in the muscles of the arms and legs, girdle sensations about the chest and mild muscular twitchings may precede a major attack. One of the earliest signs is that of Chvostek—a twitching of the facial muscles when tapping over the course of the facial nerve. Other early signs are a circumoral pallor and nervous excitement and apprehension occasionally bordering on hysteria. In severe tetany a characteristic sign is carpopedal spasm—a flexion of the fingers at the metacarpophalangeal joints with adduction of the thumb and turning inward of the foot at the ankle joint with adduction of the great toe. Muscle cramps are common and the voluntary muscles may undergo clonic or tonic convulsions. When the glottis is involved, breathing may become labored and noisy. Severe cyanosis and cessation of respiration sometimes occur as a result of paresis of the intercostal muscles and diaphragm. Severe generalized convulsions may lead to sudden death. The severity of the symptoms parallels the fall of the blood calcium which normally averages 10 mg. per cent. Ordinarily a level of 7 mg. per cent or less produces symptoms. The prognosis depends upon the extent of the damage to the parathyroids. Complete removal of all four parathyroids is of course incompatible with life. When the parathyroids have been extensively traumatized or their delicate nutrient vessels have been thrombosed, they act as glandular transplants and treatment should be continued until their circulation has become reestablished and normal secretory activity has returned. In mild transient cases, a diet liberal in milk and cheese, augmented by calcium salts by mouth or intravenously and vitamin D is generally sufficient to tide the patient over. In severe cases, periodic parathormone injections may be required. The type and extent of treatment should be controlled by frequent blood-calcium determinations.

In our experience, tetany has occurred only sporadically and for the most part disappeared with routine treatment usually before the patient's discharge from the hospital. In recent years A. T. 10 has supplanted parathormone in protracted cases. However, in our clinic we have not found it necessary to resort to this potent drug. We attribute our low incidence of tetany to: (1) The fact that we do not practice ligation of the inferior thyroid arteries. (2) Meticulous care in avoiding deep suture ligatures when controlling bleeding in the

tracheolaryngeal angle. (3) Avoidance of the posterior danger area of Crotti.

RECURRENCES

Adenoma. Continued and recurrent hyperthyroidism following operation for adenoma undoubtedly result from the failure to recognize and remove all the nodules particularly when dealing with the younger patients. If it were possible in all cases to remove all the adenomatous tissue and leave all the normal tissue, recurrences would practically be reduced to nil.

Hyperplasia. In general it is held that recurrences following bilateral resections are due principally to failure to remove adequate amounts of hyperplastic tissue. While this is generally true, it is by no means invariably so. The problem cannot be reduced to formula. Every case must be judged upon its own peculiar merits and in the light of surgical experience. However, when the surgeon has weighed the various criteria—the duration and severity of the clinical picture, the behavior of the basal metabolic rate before and after iodine medication, the character and consistency of the gland—he may do a radical resection comparable to that employed with success in similar cases only to find a possible persisting, mild hyperthyroidism which in several months or years may eventuate in a return of the original clinical picture possibly even in exaggerated form. Moreover, he may be annoyed to find that the original remnants have hyperplased to such a degree that the recurrent goiter is actually larger than the original.

It is important that no detached remnants of upper pole be allowed to remain in proximity to the superior thyroid artery lest they act subsequently as foci for further hyperplasia and growth. Likewise, the resection of a pyramidal lobe, even though diminutive, is important. An appreciable number of recurrences have been traced to this source. When dealing with certain cases of fulminating hyperplasia that show little or no iodine resolution, we occasionally deliberately court a transient postoperative hypothyroidism particularly in the younger age groups. (Fig. 12.)

Inasmuch as the etiological factors of goiter are very complex and doubtless multiple, it is probable that many recurrences are based on the original cause or "urge" which, while temporarily subdued by thyroidectomy, may still be lying dormant. Thus to be completely effectual and curative, thyroidectomy besides reducing the thyroid gland should break the chain of causative factors—adrenal, thyroid,

pituitary and sympathetic nervous system. In a certain number of cases, this cycle apparently is not broken and produces recurrences.

MORTALITY IN GOITER OPERATIONS

In the ten-year period, 1919 to 1929 inclusive, 1755 operations were performed upon 1553 patients with hyperthyroidism at the Long Island College Hospital.[13] There were twenty-two fatalities—an operative mortality of 1.2 per cent.

In the ten-year period, 1929 to 1939 inclusive, 3426 operations were performed upon 3347 patients. There were thirty fatalities—an operative mortality of .86+ per cent which compares favorably with the 1.2 per cent mortality for the first ten years.

Combining these results, we find that in the twenty-year period, 1919 to 1939 inclusive, there were fifty-two deaths following 5181 operations upon 4900 patients with hyperthyroidism in the Goiter Clinic of the Long Island College Hospital—an operative mortality of .98+ per cent. Of these deaths twenty-eight were in instances of exophthalmic goiter, twenty-two in adenoma and two in carcinoma.

Causes of Death (1919 to 1939 Inclusive)	Fatalities
Postoperative hyperthyroidism (crisis) and secondary cardiac failure	19
Primary cardiac failure	12
Embolism	6
Pneumonia	5
Coronary disease, ventricular fibrillation	3
Infection, sepsis, septicemia	3
Respiratory failure	2
Intravenous injection (accident)	1
Tetany	1
	52

Postoperative crisis still remains the greatest factor in mortality. It accounted for nineteen deaths in our series. Stage procedures may lessen the likelihood of crisis but they by no means entirely eliminate the danger. We have had fatalities following a single ligation and following second and even third-stage procedures. Several cases of fatal crisis followed operation in elderly people for the removal of large, relatively non-toxic adenomas with little or no increase in the basal metabolic rate. Crisis seems thus not to be limited to active hyperplasias with increased metabolism.

Primary heart failure was responsible for twelve deaths. These patients entered the hospital for the most part through the Out-Patient Department and had been suffering for many years from chronic hyperthyroidism with degenerative effects—myocardial in-

sufficiency, decompensation, hypertension, diabetes and other conditions of bad prognostic significance. They were largely desperate risks.

Embolism caused six fatalities. The patients were predisposed by chronic endocarditis, arteriosclerosis, myocardial insufficiency and senility and were, therefore, known to have guarded prognoses.

Pneumonia accounted for five fatalities in rather bad risk patients, yielding in a series of 5181 operations a percentage of only .09+ per cent.

Coronary disease and ventricular fibrillation accounted for three sudden deaths in patients predisposed by serious damage to their cardiovascular systems—chronic fibrillation, cardiac hypertrophy and arteriosclerosis.

Sepsis, respiratory failure, tetany and an accident connected with a postoperative intravenous injection of glucose accounted for the remaining six fatalities.

It is not within the scope of this report to review in great detail the various factors leading to increased mortality. However, irrespective of certain conditions inherent in the individual patient and over which we have only limited control, we believe that the abuse of iodine in the so-called medical treatment strongly predisposes to increased mortality and that we can and should control this factor. When it is more generally recognized that iodine has no place in the medical treatment of hyperthyroidism, we believe that mortality statistics will be further reduced. Other surgeons have likewise emphasized this matter.[14] Twenty-four of fifty-two fatal cases in our series were known to have had indiscriminate treatment with iodine. These included practically all of those dying in crisis. In conclusion, we are of the opinion that if iodine therapy were limited to the standardized preoperative preparation of the hyperthyroid patient, the incidence of dreaded crisis could be reduced to practically nil.

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The American Journal of Surgery

PUBLISHED MONTHLY BY THE AMERICAN JOURNAL OF SURGERY, INC.
49 WEST 45TH STREET, NEW YORK, N. Y.

Editor: THURSTON SCOTT WELTON, M.D., F.A.C.S., NEW YORK

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NEW SERIES, VOLUME XLIX

JULY TO SEPTEMBER

1940

THE AMERICAN JOURNAL OF SURGERY, INC., PUBLISHERS
NEW YORK MCMXL

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Printed in the United States of America

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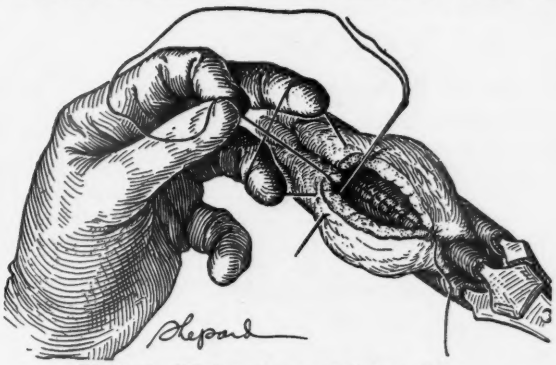

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*Campbell, W. C. and Speed, J. S., Annals of Surgery, 110-1. July, 1939. • Berry, H. C., Journal of Arkansas State Dental Association, 10-2. June, 1939. • Strock, A. E., American Journal of Orthodontics & Oral Surgery, 25-5. May, 1939. • Doherty, W. B., American Journal of Ophthalmology, 22-4. April, 1939. • Smith-Petersen, M. N., The Journal of Bone & Joint Surgery, 21-2, April, 1939. • Claflin, R. S., American Journal of Orthodontics & Oral Surgery, 25-1. January, 1939. • Hopkins, H. H. and Zuck, F. N., Medical Bulletin of Veterans' Administration, 15-3. January, 1939. • Venable, C. S. and Stuck, W. G., Journal of American Medical Association, 11-15. October 8, 1938.

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SOCIETY MEETINGS

AMERICAN ACADEMY OF ORTHOPAEDIC SURGEONS
Boston, January 21–25, 1940. (Secy.—Dr. Carl Badgley, University Hospital, Ann Arbor, Mich.)

AMERICAN ASSOCIATION FOR THE STUDY OF GOITER
Rochester, Minn., April 15–17, 1940. (Secy.—Dr. W. Blair Mosser, Kane, Penna.)

AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA
Atlantic City, June 7–8, 1940. (Secy.—Dr. Ralph G. Carothers, 409 Broadway, Cincinnati)

AMERICAN ASSOCIATION OF GENITO-URINARY SURGEONS
Skytop, Penna., June 20–22, 1940. (Secy.—Dr. Charles C. Higgins, 2020 East 93rd Street, Cleveland)

AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS
New York, June 4–7, 1940. (Secy.—Armour G. Park, 540 N. Michigan Ave. Chicago)

AMERICAN ASSOCIATION OF OBSTETRICIANS, GYNECOLOGISTS AND ABDOMINAL SURGEONS
Excelsior Springs, Mo., September 19–21, 1940. (Secy.—Dr. James R. Bloss, 418—11th St., Huntington, W. Va.

AMERICAN ASSOCIATION OF RAILWAY SURGEONS
Chicago, September 16–18, 1940. (Secy.—Dr. D. B. Moss, 547 W. Jackson Blvd., Chicago)

AMERICAN COLLEGE OF SURGEONS
Chicago, October 21–25, 1940. (Secy.—Dr. Frederic A. Besley, 215 N. Sheridan Rd., Waukegan, Ill.)

AMERICAN CONGRESS OF PHYSICAL THERAPY
Cleveland, September 2–6, 1940. (Secy.—Dr. Richard Kovacs, 2 East 88th Street, New York)

AMERICAN GYNECOLOGICAL SOCIETY
New York, June 17–19, 1940. (Secy.—Dr. Richard W. TeLinde, 11 East Chase St., Baltimore)

AMERICAN MEDICAL ASSOCIATION
New York, June 10–14, 1940. (Secy.—Dr. Olin West, 535 N. Dearborn Street, Chicago)

AMERICAN ORTHOPAEDIC ASSOCIATION
Kansas City, May 6–9, 1940. (Secy.—Dr. Ralph K. Ghormley, The Mayo Clinic, Rochester, Minn.)

AMERICAN PROCTOLOGIC SOCIETY
Richmond, June 9–11, 1940. (Secy.—Dr. Curtice Rosser, 710 Medical Arts Bldg., Dallas)

THE AMERICAN SOCIETY OF ANESTHETISTS, Inc.
New York, April 10, 1940. (Secy.—Dr. Paul M. Wood, 745 Fifth Avenue, New York)

AMERICAN SURGICAL ASSOCIATION
St. Louis, May 1–3, 1940. (Secy.—Dr. Charles G. Mixter, 319 Longwood Avenue, Boston)

ASSOCIATION OF MILITARY SURGEONS OF THE U. S.
Cleveland, October, 10–12, 1940. (Secy.—Dr. H. L. Gilchrist, Army Medical Museum, Washington, D. C.)

CONGRESS OF ANESTHETISTS
Chicago, October 21–25, 1940. (Secy.—Dr. E. Klaus, 1699 W. 25th Street, Cleveland)

INTERNATIONAL COLLEGE OF SURGEONS
Venice, Fla., February 11–14, 1940. (Secy.—Dr. Charles H. Arnold, 903–15 Terminal Bldg., Lincoln, Nebr.)

INTER-STATE POST-GRADUATE MEDICAL ASSOCIATION OF NORTH AMERICA
Cleveland, October 14–18, 1940. (Secy.—Dr. Tom B. Throckmorton, 406 Sixth Avenue, Des Moines)

NEW ENGLAND SURGICAL SOCIETY
Poland Spring, Me., September 27–28, 1940. (Secy.—Dr. John F. Gile, Hanover, New Hampshire)

PACIFIC COAST SOCIETY OF OBSTETRICS AND GYNECOLOGY
San Francisco, November 6–9, 1940. (Secy.—Dr. T. Floyd Bell, 400—29th Street, Oakland, Calif.)

PACIFIC COAST SURGICAL ASSOCIATION
Portland, April 3–6, 1940. (Secy.—Dr. H. Glenn Bell, University of California Hospital, San Francisco)

PAN-PACIFIC SURGICAL ASSOCIATION
Honolulu, 1942. (Secy.—Dr. Forrest J. Pinkerton, Young Bldg., Honolulu)

SOUTHEASTERN SURGICAL CONGRESS
Birmingham, March 11–13, 1940. (Secy.—Dr. B. T. Beasley, 701 Hurt Bldg., Atlanta)

SOUTHERN SURGICAL ASSOCIATION
Hot Springs, Va., December 10–12, 1940. (Secy.—Dr. Alton Ochsner, 1430 Tulane Avenue, New Orleans

WESTERN SURGICAL ASSOCIATION
Topeka, Kans., December 6–7, 1940. (Secy.—Dr. Albert H. Montgomery, 122 S. Michigan Blvd., Chicago)

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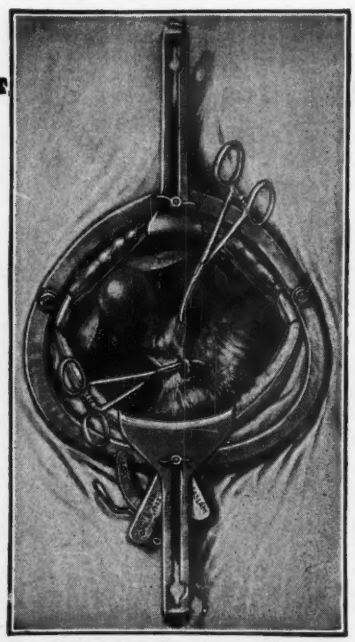

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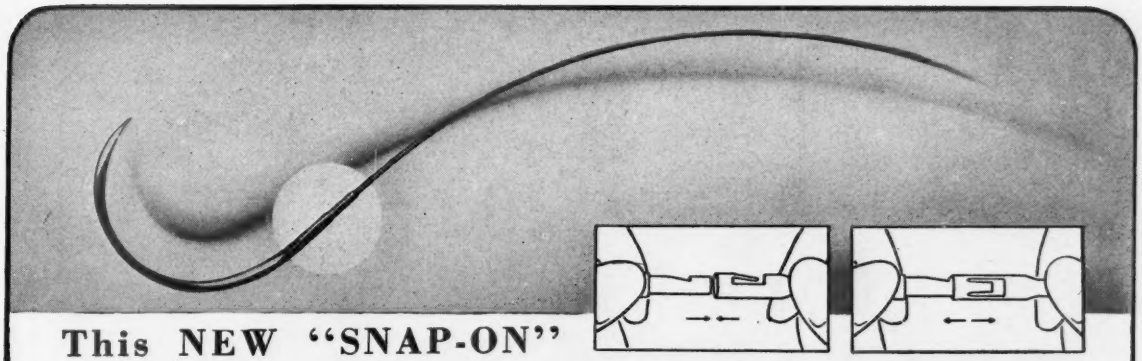

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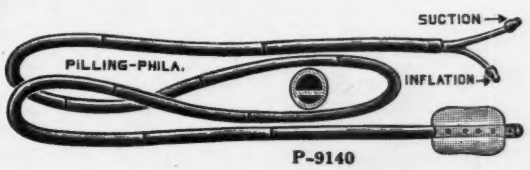

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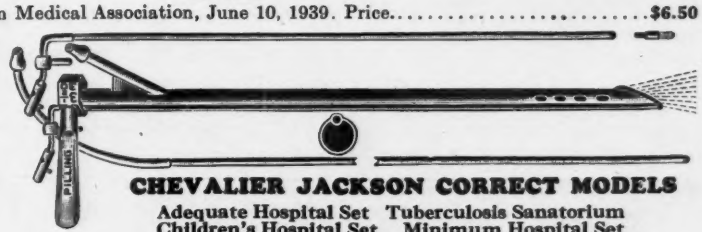

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